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ABSTRACT

One of a special series on population change and development, this paper focuses primarily on the programs and policies responsible for mortality or fertility change, rather than on the growth impact of a mortality or fertility change per se. The first portion of the document examines three models for assessing the population growth implications of mortality and fertility reduction measures. Model A, a current approach, illustrates the growth implications of changed mortality; Model B, another current approach, shows the growth implications of changed fertility; and Model C, the proposed approach, presents the growth implications of mortality or fertility change measures. Through the use of two qualitative illustrations, e.g., child survival programs and equitable development measures, the second section of the paper examines the implications of using Model C. In part three of the document three quantitative illustrations (overall development, income distribution, and child spacing) are used to assess further implications of using Model C. The paper's final part offers conclusions and discussion about the three models. Charts and tables are included to illustrate the models. Three annexes examine further the issues of overall development, income distribution, and child spacing. (RSL)

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Toward a More Relevant Assessment of the Relationships among Them

Davidson Gwatkin

WORLD BANK STAFF WORKING PAPERS

Number 686

POPULATION AND DEVELOPMENT SERIES

Number 11

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Washington, D.C., U.S.A.

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FOREWORD

This paper is one in a special series of World Bank Staff Working Papers on population change and development. Prepared as background papers for the World Development Report 1984, they provide more detailed treatment and documentation of the issues dealt with in Part II of the Report. The papers cover a range of topics, including the effects of fertility and mortality, the links between population growth and internal and international migration, and the management, financing, and effectiveness of family planning programs. They include several country and regional studies of fertility change and population policy.

The background papers draw on a large number of published and unpublished studies of individual researchers, on Bank policy analysis and research, and on reports of other organizations working on population and development programs and issues. The papers are the work of individuals and the views and interpretations expressed in them do not necessarily coincide with the views and interpretations of the Report itself.

I hope these detailed studies will supplement the World Development Report 1984 in furthering understanding of population and development issues among students and practitioners of development.

Nancy Birdsall
Staff Director
World Development Report 1984

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- Ainsworth, Martha. Family Planning Programs: The Clients' Perspective. World Bank Staff Working Paper no. 676.
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- Zachariah, K.C. The Anomaly of the Fertility Decline in India's Kerala State: A Field Investigation. World Bank Staff Working Paper no. 700.

Abstract

What are the population growth consequences of public policies and programs to reduce mortality? How large a change in growth will result from efforts to lower fertility?

An adequate response to such questions requires an approach which begins with the programs and policies under consideration and examines their overall influence on fertility and mortality. There are two types of influence to be covered: first, the influence of the policies or programs in question on mortality and on fertility; and second, the influence of the resulting fertility or mortality decline on the rate of change in the other.

Current approaches, which focus primarily on the growth consequences of a change in mortality or fertility per se, largely overlook the former of these two types of influence. This makes them inadequate for the assessment of public policies and programs, especially for the assessment of the many policies and programs undertaken for the purpose of reducing mortality or fertility which have a strong direct impact on the other as well.

An approach of the sort required is more difficult to apply than to espouse. But its potential significance for policy and program analysis can be illustrated through a few qualitative and crude quantitative examples. These illustrations, taken in conjunction with the conceptual considerations presented, suggest two propositions to serve as interim guides to action and as hypotheses to be confirmed or disproven through future research:

-- Most programs and policies undertaken to reduce mortality will, in most settings and over the long run, produce even larger fertility declines and thus slower growth.

-- Most measures designed to reduce fertility will also result in slower growth, but by an amount that may upon occasion be significantly less than anticipated on the basis on conventional assessment techniques.

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Condensé

Quelles sont, pour la croissance démographique, les conséquences des politiques et programmes lancés par les gouvernements pour réduire la mortalité? De quelle ampleur peut être la variation de la croissance à attendre des efforts en vue de réduire la fécondité?

Pour obtenir une réponse satisfaisante à ces questions, il faut une méthode qui parte des programmes et politiques examinés et qui étudie leur influence générale sur la fécondité et la mortalité. Deux types d'influence sont à considérer : premièrement, l'influence des politiques ou programmes en question sur la mortalité et sur la fécondité; et deuxièmement, l'influence de la baisse de la fécondité ainsi suscitée sur le taux de variation de la mortalité, et vice versa.

Les méthodes actuelles, qui se concentrent au premier chef sur les conséquences pour la croissance d'une variation de la mortalité ou de la fécondité uniquement, négligent dans l'ensemble le premier des deux types d'influence précités. De ce fait, elles ne sauraient convenir à l'évaluation des politiques et programmes gouvernementaux, notamment à l'évaluation des nombreux programmes et politiques lancés pour réduire la mortalité qui ont également une forte incidence sur la fécondité, et vice versa.

Une méthode du type nécessaire est plus difficile à appliquer qu'à adopter. Toutefois, on peut illustrer son importance potentielle pour l'analyse des politiques et des programmes en donnant quelques exemples qualitatifs et quelques chiffres. Ces exemples, examinés à la lumière des considérations de principe présentées, permettent de formuler deux propositions destinées à servir de directives provisoires pour une action et d'hypothèses, qui devront être confirmées ou réfutées par des travaux de recherche futurs:

- La plupart des programmes et politiques entrepris pour réduire la mortalité déboucheront, le plus souvent et à longue échéance, sur une baisse encore plus forte de la fécondité, et par conséquent sur une croissance plus lente.

- La plupart des mesures destinées à réduire la fécondité entraîneront également un ralentissement de la croissance, mais d'une ampleur qui pourrait, à l'occasion, être sensiblement plus modeste qu'on ne l'avait prévu en se fondant sur les techniques classiques d'évaluation.

Extracto

¿Cuáles son las consecuencias para el crecimiento de la población de las políticas y programas públicos destinados a reducir la mortalidad? ¿Qué dimensiones tendrá el cambio en el crecimiento que resultará de los esfuerzos por disminuir la fecundidad?

Para responder adecuadamente a estos interrogantes se requiere un enfoque que comience con los programas y políticas que se estudian y que examine su influencia general en la fecundidad y la mortalidad. Hay que referirse a dos tipos de influencia: primero, la de las políticas y programas objeto del examen sobre la mortalidad y la fecundidad, y segundo, la influencia de la disminución resultante de la fecundidad o la mortalidad sobre la tasa de cambio en la otra.

Los enfoques actuales, que se centran primordialmente en las consecuencias en el crecimiento de un cambio en la mortalidad o la fecundidad en sí, en gran parte desatienden el primero de estos dos tipos de influencia. Esto los hace inapropiados para la evaluación de las políticas y programas públicos, en especial la evaluación de las muchas políticas y programas implantados con el objeto de reducir la mortalidad, o la fecundidad, que también tienen fuertes repercusiones directas en la otra.

Un enfoque del tipo que se requiere es más difícil de aplicar que de apoyar y defender. Pero su posible importancia para el análisis de políticas y programas puede ilustrarse mediante unos pocos ejemplos cualitativos y cuantitativos básicos. Estas ilustraciones, tomadas en conjunto con las consideraciones conceptuales que se presentan, sugieren dos proposiciones que servirán de orientaciones provisionales en la adopción de medidas y como hipótesis que se confirmarán o se refutarán mediante futuras investigaciones:

-- La mayoría de los programas y políticas emprendidos para reducir la mortalidad, en la mayoría de los ambientes y a largo plazo producirán disminuciones aún mayores de la fecundidad y, por lo tanto, un crecimiento más lento.

-- La mayor parte de las medidas destinadas a reducir la fecundidad también darán por resultado un crecimiento más lento, pero en un porcentaje que en ocasiones puede ser considerablemente menor de lo previsto según técnicas de evaluación corrientes.

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Introduction

A great deal of work has gone into exploring the influence of mortality reduction on fertility and on population growth. A comparable amount of effort has been devoted to demonstrating the population growth implications of faster fertility decline.

The quality of this research has often been impressive, and its findings have frequently been illuminating. Its results, however, can be misleading. For the population growth consequences of a mortality or fertility change per se are not necessarily the same as — and might well, under some circumstances, be quite different from — the population growth implications of the policy or program producing that change, because of other demographic effects produced by the policy and program in question.

What matters from a public policy perspective is the overall impact of measures available for consideration or under implementation rather than the effects of only one of the changes resulting from them. An adequate appreciation of the population growth implications of public efforts will thus require movement away from the present focus on the growth impact of a mortality or fertility change per se, toward a broader assessment that starts with the programs and policies responsible for that mortality or fertility change.

What follows represents an initial effort in this direction. It is to proceed in the three stages. The first will feature a presentation of the

approach to be advanced, along with an explanation of how it differs from and why it appears preferable to those currently in use. The second stage will consist of a discussion of two prominent recent policy and program proposals -- one for reducing fertility, the other for reducing mortality -- a discussion designed to show how an assessment of the proposals' population growth implications would be affected by using the approach being advanced in place of those currently in use. Third will come an extension of the qualitative presentation of part two by means of a few elementary, crude quantitative illustrations. This will be followed by a brief summary and word of encouragement to anyone interested in undertaking the much more careful quantitative work that will be required if the full demographic impacts of fertility and mortality measures are to be adequately appreciated.

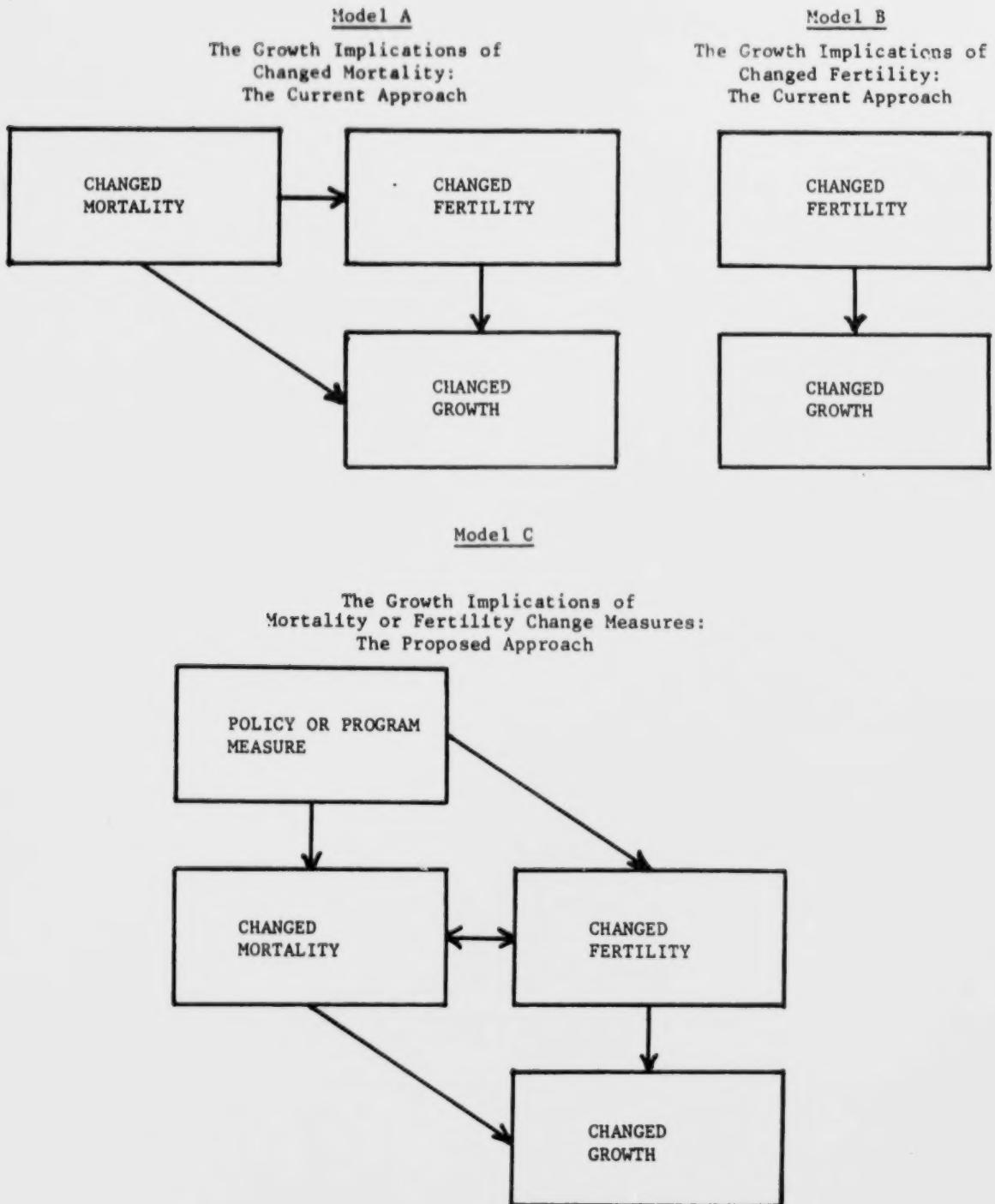
I. Three Models for Assessing the Population Growth Implications of Mortality and Fertility Reduction Measures

The need for change in approach and the nature of the changes needed can be illustrated with reference to the three simple models whose principal features are sketched in figure 1: models A, B, and C.^{1/} Models A and B are those currently in use: model A with respect to mortality reduction, model B with respect to fertility reduction. Model C is that to be advanced as more appropriate.

The Models in Brief

Model A has the initial decline in mortality as its starting point. According to it, mortality decline affects population growth in two ways. First, the reduction in mortality operates directly on population growth: ceteris paribus, the larger the mortality decline, the greater the accelera-

Figure 1: Approaches to the Assessment of Demographic Change



tion of growth. Second, it works indirectly through fertility. Mortality decline, especially infant mortality decline, produces a reduction in fertility by extending the period of lactation and by reducing the number of births required for a couple to produce a given number of surviving offspring. This fertility decline, which tends to reduce population growth, represents a countervailing pressure to that produced by the mortality decline's more direct growth influence.

The amount of work being undertaken within the framework of this model is voluminous. Of special concern has been a delineation and assessment of the mechanisms through which infant mortality decline leads to a reduction of fertility: the separation of the physiological from the behavioral; the division of the behavioral into "replacement" and "insurance" or "hoarding" effects; the measurement of the quantitative significance of each. Of equal interest to researchers has been the overall size of the indirect relative to the direct influences of mortality decline on population growth: whether, in particular, the influence of infant mortality reductions on fertility is large enough to offset fully the pressure for faster population growth those reductions also generate.^{2/}

Model B -- if the term "model" can appropriately be applied to something so simple as two boxes connected by an arrow -- is even more straightforward than model A. It begins with fertility decline, which is translated directly into slower population growth. Model B is simpler than model A because it lacks provision for an indirect effect on population growth via mortality reduction. The implicit assumption is that faster fertility decline does not affect the rate of mortality decline.

This is the model behind the standard approach used in hundreds of population projection exercises undertaken to demonstrate the influence of faster fertility decline: exercises featuring variations in the rate of fertility decline while leaving the rate of mortality decline unchanged in order to produce alternate projections of population size whose differences are attributable to differences in the path of fertility alone.^{3/} This approach has also been taken in presenting the results produced by many well-known demographic-economic models designed to demonstrate the influence of faster fertility decline and slower population growth on economic output.^{4/}

As abstractions, both models A and B are quite valid. And as such, they have proven to be of great value in pointing to features of demographic change that would not otherwise be intuitively obvious.^{5/}

They are not, however, adequate for policy or program analysis. They would be satisfactory for this purpose only if mortality and fertility declines could be made to happen exogenously -- if policy makers could simply wish them into being. But this is not possible. Those who desire changes must put into effect policies and programs to produce them; and these policies and programs have demographic consequences that can be considerably wider than those captured by models A and B. And this means results that could be correspondingly different.

Thus model C, designed to capture these other consequences. As can be seen, both models A and B are contained in it, but it is broader than either. In particular, model C starts with a policy, program, or other development leading to a change in mortality and/or fertility, rather than with the mortality or fertility change itself. And it examines simultaneously the mor-

tality and fertility effects of the policy, program, or development under examination in assessing its significance for growth.^{6/}

The Models and Mortality Reduction Measures

Model C provides two channels whereby activities undertaken for the purpose of reducing mortality can influence fertility. The indirect channel of model C, which operates via the mortality reduction produced, is the same as the single channel of model A. But in addition to the indirect channel, model C incorporates a further, largely overlooked direct channel^{7/} whereby mortality reduction policies and programs can influence fertility by inducing changes in attitudes and behavior in ways that are largely independent of whatever mortality reduction might be achieved.

This second channel is incorporated in recognition of the importance of the direct fertility reduction influence exercised by many measures frequently discussed in terms of their mortality impact.^{8/} An example is women's education, which has been closely linked with reductions in infant mortality.^{9/} For every point of infant mortality reduction attributable to improved maternal education, there would be the indirect impact on fertility described above with respect to both models A and C, an impact presumably equal in magnitude to that produced by a comparably large infant mortality reduction resulting from any other measure. But, as is well known, women's education can also affect fertility directly: by delaying marriage to permit the completion of schooling, for example; through increasing women's qualifications for jobs outside the home that can compete with child-bearing as a means of gaining satisfaction and status; by enhancing the likelihood that the lower family size ideals of younger women will prevail over those of older generations.^{10/}

Such effects would produce additional downward pressure on birth rates that would be ignored were only the indirect influence to be considered.

To say that a direct influence can exist is not, of course, to claim that it will inevitably be important. At least some mortality reduction measures that have proven effective in the past and that are frequently mentioned as possible models for the future seem likely to exercise little if any direct impact on fertility. Examples include quasi-military approaches to malaria reduction through the application of pesticides by spraying teams from outside the villages affected, and the somewhat similar approach to vaccination and containment that worked so well against smallpox.^{11/} In neither case is it possible to see how the approach used might have contributed to any lasting changes in couples' attitudes or reproductive behavior other than those resulting from the mortality decline that occurred.

The strength of the direct fertility influence thus seems likely to vary greatly from measure to measure; and because of this, the use of model C implies that the magnitude of fertility decline associated with any given amount of mortality reduction is not fixed. Under model A, whose only channel of influence on fertility flows from mortality decline to fertility, there is little room for variability: a ten-point infant mortality decline is a ten-point decline, regardless of how produced. But when the possibility of a highly variable direct influence is added, the picture changes. The amount of fertility decline per unit of mortality reduction becomes a function of the measures or developments by which the mortality reduction in question is produced.

The Models and Fertility Reduction Measures

Model C also provides two channels through which measures initiated in order to reduce fertility can lower mortality as well. The first channel is direct, analogous to that discussed earlier with respect to the effect of mortality reduction measures on fertility. The second channel is indirect, permitting the fertility reduction achieved to exercise an effect on mortality decline. This effect, comparable to the much-discussed influence of mortality reduction on fertility, has been curiously ignored in quantitative assessments of fertility reduction's population growth implications despite the publicity that family planning advocates give to fertility reduction's health and mortality benefits.

The first of these two channels is intended to account for the propensity of many -- although not necessarily all^{12/} -- fertility reduction approaches to act directly on mortality as well.^{13/} An example is income distribution, whose relationship with fertility has been widely discussed.^{14/} Because of the non-linearity of the income-fertility relationship, it is often argued that a pattern of development featuring faster improvements at lower rather than at higher income levels will produce a greater impact on fertility than a pattern where the largest gains are enjoyed by the better-off. To think in terms of non-linearities with respect only to fertility, though, would obviously be a mistake. For they exist at least equally clearly if not even more clearly with respect to mortality. It is well established that the poor will spend a much larger proportion of any income increment on basic commodities relevant for health than will the rich, thus implying that income channeled to the poor will produce a commensurately greater impact on mortality.^{15/}

The second, indirect channel is designed to account for well-known change in mortality that can be produced by fertility reduction per se. Fertility reduction can be expected to result in a mortality decline when the fertility reduction occurs through particularly large declines in births with a higher-than-average risk of early death and/or through a shift of births from a high-risk to a low-risk category: that is, when older women with many children are among the earliest to practice family limitation, and when birth intervals lengthen.^{16/} Where this mortality impact is found, the population growth influence of fertility reduction will be diluted in the same manner that the mortality decline's impact on growth is lessened by the induced change in fertility.

II. The Implications of Using Model C: Two Qualitative Illustrations

So much for the theory. What does all this have to do with today's world and the way in which population policies and programs are shaped and assessed in it?

One way of responding to such a query is to supplement the brief allusions to policy implications scattered through the preceding theoretical discussion with a systematic look, through the lenses of the different models presented, at the population growth implications of some measures currently being advanced to alter mortality and fertility trends. In this manner, it is possible to get a preliminary, qualitative sense of the difference that would result from applying model C rather than model A or B.

Two sets of proposed measures are especially well suited for this illustrative purpose. One, concerned primarily with mortality, features pro-

posals for increasing child survival probabilities. The other consists of packages of development measures recommended for the reduction of fertility.

Child Survival Programs

In recent years, several international agencies have expressed interest in developing or implementing measures that would increase the likelihood of child survival. Among the best known are the World Health Organization's emphasis on primary health care, UNICEF's proposal for a "child survival revolution," and the Ford Foundation's "Child Survival/Fair Start" program.^{17/}

Since such interest arises primarily out of a desire to reduce mortality, the conventional way of assessing the population growth implications of programs emanating from it would be through the application of model A, with its focus on the fertility reduction resulting from whatever improvements in infant and child mortality might be produced.

The use of model C's broader outlook could be expected to produce results differing in two ways from those of such a conventional approach. First, the contribution of child survival programs to fertility reduction will often be stronger. Second, the magnitude of this contribution will not be fixed but will vary according to the mix of measures employed.

Because of its specificity, the UNICEF "child survival revolution" is particularly well suited to an illustration of the point. The UNICEF proposal, first advanced in late 1982, is a package of seven measures known by the acronym "GOBI-FFF."^{18/} The seven measures are:

- Growth monitoring: the use of child growth charts along with regular weighing and back-up advice to help parents and health workers identify children at risk.

- Oral rehydration therapy: the oral administration of a simple solution to counter the dehydration that is the most frequent immediate cause of death resulting from diarrhea-producing infections.

- Breast feeding: the promotion of awareness about the benefits of breast milk and continued reliance on it.

- Immunization: the expansion of programs to vaccinate against six major communicable diseases of childhood: measles, tetanus, whooping cough, diphtheria, poliomyelitis, and tuberculosis.

- Food supplements: the furnishing of extra food to poor mothers for the purpose of reducing the incidence of low birth weights, and for high-risk children at the time of weaning.

- Family spacing: the provision of the knowledge and the means mothers need to lengthen the interval between births.

- Female education: the improvement in the general knowledge base and status of the mother who represents, in UNICEF's terms, "the most important primary health care worker."^{19/}

An assessment of a GOBI-FFF program implications done in terms of the conventional (model A) framework examining only the indirect effect would in all likelihood lead to the conclusion that the fertility influence is not strong enough to offset fully the change in mortality decline. For there is a general consensus among researchers that the replacement effect of mortality decline is significantly less than one-for-one; and a strong suspicion that, at least in the short run, the addition of the other indirect effects of anti-mortality measures on fertility are not large enough to make up the difference. To the extent this is so, a GOBI-FFF program would be found to

produce at least some increase in population growth. The increase would not be large, to be sure, for demographic conditions are now such that even the fastest acceleration in infant child mortality decline realistically imaginable will produce an increment that is modest relative to that which would occur should trends continue as presently envisioned.^{20/} But however limited the difference, it would probably be found to lie in the direction of a larger rather than a smaller population size.

Such a conclusion would be distinctly less likely when the examination is conducted in the context of a broader framework, like that of model C, which can accommodate the direct as well as the indirect fertility influence of GOBI-FFF program measures. For many of those measures clearly have strong direct effects.

Female education's direct influence has already been described. Breast-feeding and extended birth intervals routinely figure in lists of fertility reduction approaches worthy of consideration.

Such other measures as immunization, oral rehydration, and growth monitoring would not seem necessarily to affect fertility so strongly: as suggested earlier, for example, smallpox was eradicated through an immunization program lacking immediately obvious direct fertility influence. Yet, as UNICEF discussions of the GOBI-FFF programs indicate and as others have argued, the success of such measures in the future seems likely to depend heavily upon the personal behavior of those most affected.^{21/} Mothers are not likely to come or bring their children for immunizations, for example, unless they are aware that disease is caused by physiological rather than by supernatural phenomena; the success of oral rehydration or growth monitoring pro-

grams will depend heavily on the mother's awareness of how these activities can contribute. To the extent this is so, the effective implementation of such measures will require extensive educational as well as technology delivery efforts; and success will depend upon women's becoming adequately conscious and broadly "empowered" to take control of many aspects of their lives, such as fertility, in addition to being able to act effectively in areas of behavior most directly relevant for immunization, growth monitoring, and oral rehydration.

This need for education may well increase the difficulty of achieving immediate mortality gains through technologically simple programs. But it also implies that any such programs which succeed will contain heavy doses of health and related education that mean the programs will exercise a much greater effect on fertility than suggested by a consideration of the programs' technological elements by themselves, pointing toward a commensurately larger amount of fertility decline per unit of mortality reduction achieved.

This means that conventional assessments which ignore such direct effects clearly overstate any contribution to faster growth that GOBI-FFF programs might make. In fact, when these direct effects are added to the indirect influence noted earlier, it seems quite possible that some, perhaps many, GOBI-FFF program configurations would exercise a notably larger impact on fertility than on mortality, thus restraining population growth.

As noted, the addition of direct effects through a more complete (model C) approach would also introduce the possibility of a variable mortality-fertility relationship, rather than the fixed relationship suggested by conventional (model A) thinking. As a result, the relative size of the fertility

and mortality reductions produced by a program of the sort advocated by UNICEF would depend on the program's structure and implementation.

When only the fertility impact of mortality decline per se is considered, there is, as argued earlier, no basis for believing that the amount of fertility reduction associated with a given mortality decline might vary from measure to measure. Under an assessment based solely on the indirect fertility effect of mortality reduction measures, it would be reasonable to expect roughly the same population growth impact from a given amount of infant mortality reduction, regardless of whether that reduction were produced by, say, a GOBI, an FFF, or a total GOBI-FFF effort.

This would no longer be the case when direct effects are also considered. For, as just described, there are grounds for suspecting that the direct fertility effect of the GOBI-FFF program efforts can vary greatly, depending both on the mix of components and upon how the selected mix of components is implemented (whether, for example, immunization programs are introduced more as technology delivery or as education efforts). If this is so, the population growth implications of, say, a GOBI program could well differ from those of some other mix of GOBI-FF program components — such as a "FIGO," "BIFFO," or some other such effort. This variability provides program designers with an opportunity to create component mixes that will vary not only with respect to their effectiveness in reducing mortality but also respect to the ratio of fertility to mortality reduction achieved and thus with respect to the amount of change in population size produced.

Equitable Development Measures

A prominent feature of population discussions during the 1970s was attention to the demographic implications of faster and more equitable development.

Particular interest was expressed in development's fertility effects, an interest that produced material ranging in tone from the thoughtful presentations of such authors as James Kocher, Robert McNamara, and William Rich to the strident claims that "development is the best contraceptive" expressed at the 1974 Bucharest World Population Conference.^{22/} For all the important differences in tone, however, such discussions generally led toward the common conclusion that more rapid and more egalitarian development could produce faster fertility decline.

The amount of reduction in population growth that could be expected to result from this faster fertility decline was rarely if ever explicitly considered. The absence of such a consideration makes the views of those emphasizing development's importance for fertility reduction difficult to assess with confidence; but they appear to have assumed that faster fertility reduction could reasonably be expected to result in a roughly commensurate^{23/} slowing of population growth.

This, certainly, is the conclusion that would follow from applying the conventional (model B) approach to an assessment of the population growth implications of faster fertility decline resulting from development measures, for this approach does not permit consideration of the possibility that development might also produce faster mortality reduction. In allowing for the appearance of such a countervailing change, a more complete (model C) assessment would be likely to result in a more restrained conclusion about the amount of growth reduction that would result from measures which reduce fertility. On a priori grounds, in fact, there would be little basis for dismissing the possibility that at least some fertility-reducing development

measures might actually lead toward faster growth by producing an even larger decline in mortality than in fertility.

The point can best be understood with reference to a specific example. Of those available, the most concrete and thus the most suitable for illustrative purposes in the proposal of Robert McNamara contained in his well-known 1977 MIT speech.^{24/}

McNamara's principal concern, he made quite clear at the outset of his speech, was the threat posed by population growth. "Short of thermonuclear war...", he said, "it (population growth) is the gravest issue the world faces over the decade immediately ahead. Indeed, in many ways rampant population growth is an even more dangerous and subtle threat to the world than thermonuclear war, for it is intrinsically less subject to rational safeguards, and less amenable to organized control."^{25/}

His prescription for the humane reduction in growth that he sought emphasized the achievement of faster fertility decline, especially through the social and economic approaches needed to encourage couples to desire smaller families and thus to avail themselves of the contraceptive services whose provision he also advocated. In particular, McNamara proposed a five-point program. "Governments," he said, "should try to:

"— Reduce current infant and child mortality rates sharply.

"— Expand basic education and increase the proportion of girls in school.

"— Increase the productivity of smallholders in the rural areas, and expand earning opportunities in the cities for low-income groups.

"— Put greater stress on more equitable distribution of income and services in the drive for greater economic growth.

"— And above all else, raise the status of women socially, economically, and politically."26/

How much of a decline in growth would result from the implementation of such measures? The answer, which McNamara did not try to provide, will almost certainly vary according to the way in which the measures' impact is assessed.

As has been seen, any judgment framed in terms of the (model B) approach normally applied to a proposal presented as an approach to fertility reduction would involve a direct translation of any fertility reduction achieved into a slowing of population growth. For the assumption is that the measures will not change the rate of mortality decline.27/

This, however, can hardly be considered a realistic way to deal with a set of measures which, in different surroundings, could just as well be presented as a program for the reduction of mortality. Its first component is an explicit call to reduce infant and child mortality; and two others deal with the education and status of women, whose direct influence on infant mortality is well known. Clearly, no measures of this sort can reasonably be expected to produce changes in birth rates unless death rates are also reduced.

A successful application of such measures would clearly make the world a far better place than it now is, and the measures are all the more attractive because of their ability to produce mortality as well as fertility benefits. The fact that mortality as well as fertility will decline more rapidly, however, also means that any population growth reduction would be at least somewhat, perhaps significantly, smaller than suggested by an assessment assuming no change in the rate of mortality decline.

In other words, because of the inability of the standard, model B approach to accomodate such changes in mortality — whether resulting directly

from the measures in question or indirectly via the fertility changes those measures produce -- its use would project a larger reduction in population growth than the McNamara proposal would be likely to produce. Because model C explicitly takes mortality change into account, a demographic evaluation undertaken within the framework suggested by it could be expected to present a different and more restrained set of findings.

III. The Implications of Using Model C: Three Quantitative Illustrations

The two examples just presented are, of course, qualitative rather than quantitative. Many will doubtless be reluctant to accept the value of such qualitative arguments without assurance that they are also quantitatively significant.

Such assurance is difficult to provide. Neither the statistical models nor the data currently available are well suited for the task. Even if they were, the wide range of program and policy options available and the large variations in the developing country settings where they must be applied mean that a massive volume of research would be required before any valid general statements would be justified.

It is possible, however, to present crude and oversimplified quantitative illustrations which, for all their obvious inadequacies, can contribute significantly to a sense that the application of model C to empirical data is likely to yield results that are not intuitively obvious -- and that are also often likely to differ enough from those produced by the use of the conceptually inferior models A or B to justify efforts to apply model C despite its greater complexity. Aside from their policy interest, such findings represent

a justification for the more detailed work required for assessments embodying an adequately complete, "model C" approach.

The point can be illustrated by three simple examples. Selected opportunistically on the basis of the ready availability of the prior work required for the preparation of illustrations within the limited time available, they do not necessarily represent the approaches of greatest policy interest. They do, however, cover a wide range of issues. One, intended to provide a general background, deals with the pace of overall economic and social development. Another, somewhat more specific, concerns income distribution, one of the McNamara fertility proposals. The third is narrower or more specific still: child spacing, a component of the UNICEF mortality proposal.

Each example consists of a set of population projections. Each set starts with a baseline projection, developed from some more or less reasonable assumption about the movement of the determinant of mortality and fertility under consideration in the absence of special efforts to change its course. Then come projections that seek to approximate the demographic consequences of an altered rate of change in that determinant: projections based on model A, on model B, and on model C.

Overall Development

Overall development, clearly, is not a single policy that one puts into effect on any particular morning. Rather, it is the cumulative result of a myriad of actions taken over an extended period. An examination of its demographic consequences is thus still a large step from examining the demographic effects of an individual policy or program. Such an examination is nonetheless an obvious place to begin, as an initial step in the right

direction away from investigating the population growth consequences of mortality or fertility change per se toward examining the consequences of the factors that determine those changes.

Also, it is easy to produce usefully illustrative, even if far from definitive, estimates of the demographic implications of faster or slower development. These can be derived from any of several available cross-country analyses of the relationship among demographic and other social and economic variables at different stages of development,^{28/} by defining as "normal" the trajectory represented by movement through the different stages identified in such studies and by comparing the demographic implications of alternate rates of movement along this course.

Annex I presents a detailed description of how this can be accomplished for one such study: the work on the correspondence among development indicators by Donald McGranahan and his colleagues at the United Nations Research Institute for Social Development.^{29/} As explained in the annex, the approach employed consists of taking a population with the social, economic, and demographic characteristics of a typical low-income country and "moving it" at a 2% and then at a 3% annual development rate along the path described by the relationships identified by the McGranahan group. The population size associated with the 2% rate is defined as the baseline; and the population size associated with the 3% rate is estimated by applying crude approximations of models A, B, and C.

The results of the exercise are summarized in table 1. As can be seen, the differences over the short run, defined as the initial twenty-five years or so, are modest; but thereafter, they become progressively more significant. In brief:

Table 1: Differences in Population Size Associated with a 3% rather than a 2% Annual Rate of Development

Year	Population Size Associated with 3% Annual Development Scenario Relative to that Associated with 2% Scenario, in Year Indicated		
	As Assessed by Model A	As Assessed by Model B	As Assessed by Model C
0	-	-	-
25	+0.5%	-0.5%	+1.0%
50	+4.7%	-7.2%	+0.5%
75	+6.7%	-24.3%	-17.6%
100	+6.2%	-43.3%	-39.9%

Source: Annex I

Notes: See table I-3 for absolute population size figures. The figures presented above for model A are based on those in the "adjusted" column of that table.

-- Under the 3% scenario, mortality decline is much faster: over the initial 50 years, the 3% scenario produces nearly three times as large an increase in life expectancy (16.5 years vs. 5.6 years) and over twice as rapid a decline in infant mortality (76 vs. 28 points) as the 2% scenario. Application of the model A approach, with its assumption that this faster mortality decline is exogenously introduced in a manner producing no change in fertility other than that induced by the faster mortality decline itself, results in a population 6.2% larger than the baseline population after 100 years.

-- After the initial twenty-five years, during which more rapid development has little influence on the rate of fertility change, birth rates also fall faster under the 3% than under the 2% scenario. Between twenty-five and seventy-five years, for instance, the crude birth rate falls by almost twenty points under the 3 % scenario, more than double the nine points under the 2% scenario. The use of model B, which assumes an exogenously-introduced increase in fertility reduction that does not affect the rate of mortality decline, yields after 100 years a total population 43.3% smaller than that of the baseline.

-- When the population growth implications of the faster mortality decline and the faster fertility reduction are assessed together, as is done through model C, the faster fertility decline is found to be quantitatively more significant over the long run. After 25 years, the population size is slightly (1%) larger. But 100 years after the starting point of the projection, the population size under the 3% development scenario is 39.9% smaller.

For all the reasons indicated in annex I, none of these figures can be legitimately accepted as more than broadly indicative. Yet of the three alternatives, that produced by the use of model C is clearly the least flawed and thus to be preferred. For, once faster development is recognized as the common cause of the morality and fertility change under consideration, it makes no sense to consider either mortality or fertility change alone; if changes are to be introduced, the two have to be taken together.

Acceptance of this view implies a recognition of the serious error which can result from inferring that faster mortality decline necessarily means faster population growth. When exogenously introduced, it obviously does. But when caused by a set of factors that produce even faster declines in fertility, a scenario involving more rapid reductions in mortality leads to a smaller rather than a larger population size — a size which, in this example, turns out not to be much different from that estimated without regard to development's mortality effect.

Income Distribution

Exploration of a more specific element of development policy — namely, income distribution — is also possible. Like development itself, a change in the distribution of income is the product of a mix of different programs and policies; and as such, it too is a step removed from those programs and policies themselves which would be the preferred objects of study. But as a determinant of fertility and mortality and as a symbol of a particular type of development strategy, the overall demographic implications of income distribution are of interest; and because of a long tradition of empirical exploration into its demographic effects, income distribution is much easier to examine than most policies and programs.

Especially relevant is work by T.P. Dyson, C.L.G Bell, and R.H. Cassen that examines the influence of different income distribution patterns on fertility, mortality, and population growth.^{30/} Annex II provides a description of this work and of the way in which it can be used to illustrate quantitatively the implications of selecting model A, B, or C to assess the population growth implications of fertility and mortality changes.

As indicated in the annex, the Dyson-Bell-Cassen exercise compares the demographic implications of maintaining a highly unequal pattern of income distribution as overall growth proceeds with the population growth effects of a sharp shift toward a more egalitarian pattern of distribution when the rate of overall income growth is the same. This is done through a simulation model projecting the population of low, medium, and high income groups on the basis of parameters for the relationship between mortality and income and between fertility and income established through cross-national and individual country time series econometric analyses. These analyses produce trajectories in which mortality and fertility generally decline at differing rates in response to income increases.

Table 2 presents a summary of the most relevant findings resulting from a projection exercise based on the Dyson-Bell-Cassen work. The results can be seen to vary depending upon the time frame and method of assessment adopted.

In the short run, the differences in the population growth estimates produced by the three models are relatively small: at 50 years the difference between the smallest and largest figures on table 2 is only about 3%. Such differences as exist, however, suggest clearly that egalitarian development produces faster population growth than inequalitarian development. For the

**Table 2: Differences in the Population Size
Associated with More Egalitarian
rather than Less Egalitarian
Economic Development**

Year	Population Size Associated with a "Changing Shares" Income Distribution Scenario Relative to that Associated with a "Constant Shares" Scenario, in Year Indicated		
	As Assessed by Model A	As Assessed by Model B	As Assessed by Model C
0	-	-	-
25	-	+0.5%	+1.1%
50	+0.6%	+1.8%	+3.5%
75	+3.0%	-1.7%	+4.3%
100	+6.7%	-15.5%	-4.4%
125	+10.1%	-31.5%	-17.9%
150	+11.7%	-41.6%	-28.0%

Source: Annex II

Notes: See table II-2 for absolute population size figures. The figures presented above for model A are based on those in the "adjusted" column of that table.

population of the "constant shares" or inequitably developing society is smaller than the population of the "changing shares" or more equitably developing society regardless of the method used to assess the latter population's size. This is a reflection of the greater proportion of income gain accruing to the very poor, whose fertility response to income increases is limited, during the initial stages of egalitarian development from a low starting point.

Over the longer run, however, the picture changes. The magnitude of the population size differences becomes much larger, and the way in which they are assessed exercises a much greater influence on the policy conclusions drawn:

-- An assessment focussing only on mortality change results in a larger population size because of the tendency of an egalitarian pattern to produce faster mortality decline, a result of the previously noted propensity of the poor to spend a higher proportion of an income increment on basic needs. According to the Dyson-Bell-Cassen exercise, life expectancy is 2.3 years higher after 50 years under the "changing shares" than under the "constant shares" scenario; after 100 years, the difference is 7.6 years. If this increase were to be considered an exogenous one, as in model A, the population would be 11.7% larger than the baseline population after 150 years.

-- A judgement based only on fertility change, taken as exogenously produced, obviously produces distinctly different results. Initially, for the reason discussed earlier, a "changing shares" scenario results in slower overall fertility decline than a "constant shares" scenario. After about 50 years, however, fertility falls more rapidly when shares are changing; and at 100 years the "changing shares" gross reproduction rate of

1.36 is well below the "constant shares" rate of 2.03. If only the effect of this fertility difference is considered, as in model B, the result is a much lower population total than that of the baseline: 41.6% smaller at 150 years.

-- When the faster mortality and fertility declines are considered together, through an approach like that suggested by model C, fertility once again proves stronger than mortality in determining population size. At 150 years, the "changing shares" population is 28.0% smaller than the "constant shares" baseline population. This "model C" result is also quite different from the "model A" and "model B" figures, being only about two-thirds as large as the former and nearly one-quarter larger than the latter.

If the model C results are taken to be as the least imperfect of the three, the figures produced by the conventional approaches of A and B lead are significantly misleading. Were income distribution's long-term direct effects on fertility to be overlooked, then the conclusion could legitimately be that a more equitable pattern of development would produce faster growth; whereas in fact the opposite is much more likely to be true over the longer run. To look only at distribution's fertility change implications would be to overstate by a noticeable degree the amount of slower growth that might reasonably be expected.

Child Spacing

While the long-term demographic benefits produced by faster and more equitable development may be large, the profundity of the policy shifts required to realize them is understandably unsettling to the policy maker wondering what specifically might be tried. What about something less all-encompassing, more "doable?"

Of the several candidates that come to mind, that most readily available for study is child spacing, a point in the UNICEF GOBI-FFF program and closely related to the increased breast-feeding that is another GOBI-FFF program element. Like the other determinants discussed thus far, the lengthening of birth intervals is not a program or policy in itself, but rather the outcome of efforts to extend postpartum amenorrhea by such policies as promoting breast feeding through educational measures, limiting in access to breast milk substitutes, and providing creches for working mothers; and of activities that delay conception after the reestablishment of ovulation, such as increasing contraceptive use by younger couples.^{31/} Extended birth intervals can, though, be seen as the possible sum of the impacts of a package of such measures and thus help serve as a basis for helping decide whether the benefits of such a package are likely to be large enough to justify the time and effort to develop it.

To embark on a study of child spacing's demographic impact is to enter the erudite field of birth interval dynamics, an area of demography replete with subtle ramifications that can be overlooked only at the investigator's peril. For those willing to be thought simple-minded, however, interesting suggestions about the possible demographic impact of longer birth intervals can be gained from a set of population projections featuring a change in the mother's age at each birth after the first.

Annex III provides a description of an exercise of this sort designed to illustrate the long-term effects of lengthening of the average birth interval by six month, from 24 to 30 months, within a brief period. The baseline projection of the exercise is based on parameters similar to those of the U.N.

medium variant for a typical developing country, except for the age-specific fertility schedule which is artificially constructed in a manner that provides for births at precise 24-month intervals. In the alternate projections, these parameters are altered in order to illustrate the effects of shifting from 24- to 30-month intervals. In the alternate mortality variant, infant and child mortality rates are set 15% below those of the baseline. In the alternate fertility variant, the age-specific schedule is changed in a way that lengthens the inter-generational gap by six months; and the total fertility rate is made to decline somewhat more rapidly in response to the lengthened intervals.

The results of these changes, presented in Table 3, may be summarized briefly:

-- An examination of only the 15% infant and child mortality reduction, as if it were exogenously produced, would conclude that child spacing would result in a marginally larger population size. At stabilization, for instance, the population identified by a "model A" assessment is 0.9% larger than the baseline. The small size of the difference illustrates the point made earlier about the limited population growth implications of even a rapid and significant decrease in infant and child mortality, even when the fertility effects of the measures producing that mortality reduction are not taken fully into account. Such difference as there is, though, is in the direction of a larger population, as dictated by the arithmetic and underlying logic of model A.

-- A "model B" estimate, looking only at the more rapidly fertility decline associated with longer birth intervals, would find a 5.2%

Table 3: Differences in Population Size Associated with a 30-Month rather than a 24-Month Average Interval between Births

Year	Population Size Associated with a "30-Month" Fertility Scenario Relative to that Associated with a "24-Month" Scenario, in Year Indicated		
	As Assessed by Model A	As Assessed by Model B	As Assessed by Model C
0	-	-	-
25	+0.4%	-2.7%	-1.9%
50	+0.6%	-4.1%	-2.9%
75	+0.8%	-5.0%	-3.5%
100	+0.9%	-5.2%	-3.7%

Source: Annex III

Notes: See table III-2 for absolute population size figures. The figures presented above for model A are based on those in the "adjusted" column from that period; the fertility variant used in producing the model B and model C figures incorporates both the inter-generational and total fertility rate effects of longer birth intervals. Year 100 figures refer to stable populations.

smaller population size attributable to spacing. As can be seen from the tables of annex III, the longer inter-generational gap alone would produce a population size about 1.8% smaller than that of the baseline. The remaining, larger part of the difference is attributable to the smaller total number of infants born to couples who space the births of their children. If birth spacing is to have a significant effect on fertility, in other words, much of the change will result from the change in total family size associated with spacing rather than from the spacing per se.

-- The ultimate population size estimated by accounting for both the mortality and fertility change, as under model C, would be 3.7% smaller than that of the baseline. As in each of the two other examples, the faster fertility reduction would prove to be a stronger determinant of population size than the more rapid decline in mortality^{32/}

Conclusions

The three examples just presented obviously cannot be said to represent anything like the last word on the relationship between mortality and fertility reduction efforts on the one hand and population growth on the other. As has been said before, they are methodologically crude; and by dealing with a sample of approaches that is not necessarily representative of those available, they may well leave an impression that the different models produce widely divergent results more frequently than they actually do.

The examples and the other material presented earlier, however, should when taken together at least suffice to create uneasiness about automatically continuing to follow the present approaches that feature separate consideration of only the population growth influence of mortality and of fer-

tility changes per se. With luck, the examples presented will also provoke thought about how some of the shortcomings of the analytical techniques used in them might be overcome in future research efforts.

Such efforts are clearly needed, for reasons that go far beyond the conceptual and methodological. For, notwithstanding its inadequacies, the evidence available gives rise to strong suspicions that the shortcomings of the current approaches are producing results whose biased nature is, if not inevitably, at least frequently leading to incorrect policy conclusions.

These suspicions concern the accuracy with which the demographic consequences of both mortality and fertility reduction programs are assessed. They can usefully be stated in closing, as two propositions intended to serve both as interim guides to action and as hypotheses to be confirmed or disproven through future research.

Proposition one is that when both their direct and indirect effects on mortality and fertility are appropriately taken into account, most policies and programs undertaken for the purpose of reducing mortality will in most settings and over the long run produce greater fertility than mortality declines and thus slower population growth and a smaller population size than would otherwise occur.

The proposition maintains, in other words, that the currently-prevailing view that mortality reduction will normally lead to faster long-term growth is incorrect, an artifact of inadequate model specification. This proposition is based on two considerations which, if correct, mean that the normal overall outcome of mortality reduction measures will be a reduction in long-term growth ranging in magnitude from modest to quite large.

The first consideration is that, as has been seen, the additional growth potential inherent in faster mortality reduction is normally rather small even when no allowance at all is made for the fertility effects of mortality reduction efforts, very small when a reasonable (less than fully offsetting) adjustment is entered to account for these effects. Thus, the strength of a direct fertility reduction effect needed to "tip the balance" from faster to slower growth would be modest in most cases. Even the immediate virtual elimination of infant mortality, for example, would in a typical developing country produce a stable population no more than 5% or so above that produced by a normal progression; and as can be seen from the material on child-spacing presented in annex III, even the modest fertility change represented by the inter-generational effects of a six-month extension of birth intervals is adequate to offset fully a 15% reduction in infant and child mortality.

The second consideration is that most effective mortality reduction efforts could be expected to exercise at least some direct effect on fertility. In some cases, especially those involving social and economic measures like the "general development" and "income distribution" cases discussed above, this direct effect is likely to be very large and the resulting restraint on future population expansion quite dramatic. In others, such as targetted immunization programs, it might be much smaller; and there will no doubt be at least be some instances where it will be negligible or non-existent, in which the modest increases in growth suggested by model A might well result. But such instances seem likely to be infrequent. For, as noted, there are persuasive reasons for suspecting that practically all mortality measures most currently under development will require at least some behav-

loral change on the part of those to be served if the measures are to be effective; and this will require a general awakening that will have "spill-over" or direct effects on fertility that are independent of whatever mortality results might be produced. If this assessment is correct, then the normal overall outcome of measures that lower mortality will be a reduction in long-term growth ranging in magnitude from modest to quite large.

This should not be taken to deny the possibility that, as seen in the general development and income distribution examples presented, faster mortality decline might produce at least slightly more rapid growth during an initial period that might last as long as 50-75 years. The difference is small, however, especially in relation to the much more marked tendency toward slower growth over the longer run. Nor can the word "most" safely be overlooked in reading the proposition. Its presence is meant to indicate that there will almost certainly be at least some exceptions; and it is conceivable that at least some of these exceptions^{33/} might prove significant.

Proposition two is that while not all programs and policies undertaken in order to reduce fertility will produce slower growth in the near term, over the longer run most will, when both their direct and indirect effects on fertility and mortality are appropriately taken into account, be found to result in a reduction of population growth by an amount that is at least slightly and sometimes significantly less than suggested by current assessments which fail to account for the mortality effects of those policies and programs.

As noted, two of the three quantitative examples given suggest that measures often advocated for the reduction of fertility -- faster development and more equitable income distribution -- produce modestly faster growth

during the initial fifty years or so after their introduction. The differences are not large; but this result, a reflection of the greater response to mortality to improvements in living standards during the initial stages of development from a low base, must nonetheless serve as a warning to anyone tempted to conclude from development's tendency to produce lower fertility that development also represents an immediate solution to growth problems.

At the same time, the outcomes of the quantitative examples point to longer-term differences that are much larger and thus correspondingly more impressive in the direction of a smaller rather than a larger population. Thus, the second proposition does not go as far as to claim that the conventional approaches are wrong in suggesting that fertility reduction measures will eventually produce slower rather than faster population growth. The claim is simply that these conventional approaches overstate the amount of slowing that will occur by failing to account for the tendency of fertility reducing measures to produce partly offsetting mortality declines as well.

The reason for doubting that the mortality declines will often be fully or more-than-fully offsetting over the long term is one of those mentioned earlier with respect to the first proposition: namely, an appreciation of the limited additional population growth likely to result from even sharply faster mortality reductions in most settings. This potential is so much less than that of fertility reduction that it is difficult to imagine many circumstances in which the mortality reduction produced in the process of limiting fertility would overpower the fertility effect altogether and lead to faster growth. The partial effect might still be important, however, as indicated by the income distribution example given, where a failure to account for mortality effects led to a misstatement of the likely population size by almost 25%.

Annex I: Overall Development

The work of Donald McGranahan and his colleagues, from which the overall development illustration is derived, is based on a holistic view of development. According to their approach, development is a set of interrelated social and economic changes, among which are changes in fertility and mortality. Although the processes that produce such changes may be too complex to permit the clear establishment of causal sequences, their outcomes can frequently be observed and measured; and this is what McGranahan and his colleagues seek to do.

An example of their findings is produced in table I-1. The findings were developed through an analysis of cross-sectional, national-level data employing correspondence analysis, a statistical technique which features the establishment of symmetrical relationships among the variables covered. As can be seen, "development" means for McGranahan and his colleagues a change in over a dozen indicators in ways such that the attainment of any specified level of one implies the achievement of some corresponding level of another. Thus, for example, a society with an adult literacy rate of around 60% (that prevailing at development level 50 of table I-1) would also tend to have a per capita income of about \$300, and so forth. The corresponding crude birth rate would be around 40; life expectancy would be on the order of 58 years.

The McGranahan group did not include the crude death and population growth rates among the indicators they covered. But these may be added through the use of stable population tables, as in lines 17 and 18 of table I-1, whose figures were developed by applying the Coale-Demeny "West" family stable population relationships to the crude birth rate and life expectancy derived by McGranahan et al. The result is that, in addition to the other features just noted, a society at development level 50 would be characterized by a crude death rate slightly below 11 per 1000 population, and by a population growth rate just under 3.0% annually.

Although individual countries will clearly follow widely differing courses of development, a "normal" path can be defined for illustrative purposes as that represented by progress from the lower toward the higher levels outlined in table I-1 — the trajectory identified, in other words, through a cross-sectional examination of the current configuration of country situations with respect to the different indicators presented in the table. While the complexity of the relationships just described must thwart any effort to single out progress with respect of any one variable as the principal cause of or driving force behind changes in the others, any one can usefully serve as a "reference indicator" for the purpose of measuring the rate of development progress as defined by changes in the full set of indicators covered — recognizing that, in measuring change in terms of the reference indicator, one is not describing changes in that indicator alone but rather changes in it as a shorthand way of indicating changes in all the other indicators as well. Further, it seems safe to assume that the reason mortality and fertility indicators change as one moves from left to right across table I-1 is that the

other variables are also changed -- that, in a general sense, the changes in these other variables (and the other, unmeasured factors for which they serve as proxies) are largely responsible for the shifts in mortality and fertility observed -- just as these shifts in mortality and fertility are among the factors determining changes in the other variables on table I-1.

Acceptance of these propositions permits application of the figures of table I-1 to a simple exercise to assess the influence of faster overall development on mortality, fertility, and population growth. The exercise consists of taking a hypothetical population in a low-income country, allowing it to move at different rates along the trajectory described by the progression from low to high levels of development on table I-1, and examining the demographic effects produced. By assessing these effects in terms of models A, B, and C, it is possible to assess the quantitative significance of using one model rather than the other.

Considerations of convenience dictate the use of the ubiquitous per capita income as the reference indicator. Once it has been selected, the estimates of the World Bank can be drawn upon to assist in the choice of an appropriate starting point and a reasonable set of assumptions about the future rate of social and economic progress.

McGranahan development level 13.5, used as a starting point, enjoys no special sanctity; but its correspondence with a per capita income of \$100 makes it a pleasantly round number, and the World Bank's figures suggest that it is reasonably close to the income level of the typical low-income country. According to the Bank's most recent World Development Report, the average 1981 per capita income of the thirty-four countries listed as "low-income" was \$270. A translation of this figure into the 1970 dollars used by McGranahan and colleagues, using the World Bank's 1970-81 annual inflation rate of 11.2%, yields an income level of around \$84, not unduly below the \$100 implied by the initial point selected. (The World Bank, World Development Report 1983, p. 148.)

As for the rate of progress, the 1983 World Development Report presents 1985-95 GDP growth scenarios for the low-income countries ranging from 3.6% to 4.6% annually (unweighted average for Africa and Asia). Between 1980 and 2000, the Bank's projections envision a 1.7% average annual rate of population growth in these low-income countries, producing a range of annual per capita GDP growth scenarios of roughly 1.9% to 2.9% for the coming decade. (Ibid., pp. 27, 184.)

Such figures suggest 2% and 3% annual rates of per capita income growth as reasonable possibilities. In addition to being round figures fairly close to the World Bank estimates (heroically extrapolated to cover 100 years), use of them permits an assessment of the demographic implications of another appealingly round figure: a change of one percentage point in the growth rate (of overall development measured in terms of per capita income, albeit not in terms of the McGranahan index).

Thus, the comparison made is between the population size increase associated with development at a rate defined by a 2% average annual rise in per

capita income as a baseline; and the population increase produced by the mortality and fertility changes associated with a 3% annual rise. Table I-2 presents an abridged version of the two scenarios. As can be seen there, the 2% annual per capita income increase implies a rise from the initial \$100 to \$724 over 100 years, an increase that is one dimension of the rise in the McGranahan overall development index from 13.5 to 69.9. Among the other dimensions of this rise are a decline in the crude death rate from 19.1 to 8.9, and a reduction in the crude birth rate from 46.1 to 27.4. Under the alternate 3% scenario, the McGranahan index would rise from 13.5 to 93.0. Among other things, this would imply an increase in per capita income from \$100 to \$1922, a fall in the crude death rate from 19.1 to 9.7 (and a subsequent rise to 11.6, occurring despite continuing increases in life expectancy, as a result of the population's changing age structure), a crude birth rate decline from 46.1 to 17.0 -- and also the changes in the other demographic indicators shown in table I-2 and in the other social indicators of table I-1 not reproduced in table I-2 because of space limitations.

For each of these two scenarios, it is possible to construct fertility, mortality, and population growth assumptions for use in projections. For each five-year period, an estimate of per capita income is prepared by increasing the preceding income figure by approximately 10.4% or approximately 15.9%, the amounts representing five years of progress at a 2% and a 3% annual rate respectively. Through interpolation, the corresponding development levels, crude birth rates, and crude death rates are calculated. Subtraction of the crude death rate from the crude birth rate yields an annual population growth rate for the five-year period in question, which can be used to increase the population of the preceding interval.

As indicated in the text, the two development scenarios are used in this way as the basis for four population projections. If for convenience the fertility assumptions of the 2% baseline and the 3% scenarios are designated F_1 and F_2 , respectively, and the two mortality variants are labelled M_1 and M_2 , the four projections can be identified as: F_1M_1 (baseline), F_1M_2 (initial or "crude" model A), F_2M_1 (model B), F_2M_2 (model C).

In addition, the exercise includes an "adjusted" model A projection that reflects, as the "crude" projection does not, the widely-recognized fertility influence of faster mortality decline. The adjustment approach used is largely arbitrary and dramatically inelegant: it consists of reducing the "crude" model A population figure by one-half the difference between it and the baseline figure. The justification for this approach is essentially that the results it produces are probably no worse than those yielded by any other approach that might be reasonably considered. On the one hand, the widespread recognition that faster mortality declines affect fertility makes it difficult to avoid making at least some kind of adjustment effort. On the other, the continuing uncertainty and debate about the strength of the effect renders futile any effort to derive a scientifically defensible adjustment figure; and the absence of such a figure renders quixotic any attempt to develop the sophisticated procedure that would be theoretically preferable. Such a situation calls for a simple approach that, while carefully avoiding any claim of precision, indicates recognition that faster mortality decline produces an

increase in the rate of fertility decline of a magnitude that is not hopelessly implausible in light of current knowledge or belief.

The basis for the 50% offset figure used is hardly beyond challenge; but at the same time, such a figure is not wildly out of line with the findings of recent research, which tends to produce replacement effects ranging from negligible to 80% or so. (The well-known CICRED studies, for example, produced replacement ratios of from near zero to around 0.4; and the recent World Bank analysis of World Fertility Survey data suggested replacement ratios of from near zero to 0.8. Samuel H. Preston, "Health Programs and Population Growth," Population and Development Review, Vol. 1, No. 2 (December 1975), pp. 192-93; Susan H. Cochrane and K.C. Zachariah, "Infant and Child Mortality as Determinants of Fertility: The Policy Implications," World Bank Staff Working Paper, No. 556 (February 1983), p. 28.) The 0.5 figure used is somewhat higher than the mean figure from the replacement effect studies available, a difference meant to imply informal recognition of the existence of an additional insurance effect as well as to enhance the comprehensibility of the numbers on table I-1.

Although logic and symmetry would seem to argue for an analogous attempt to adjust the model B projection for the likely tendency of faster fertility decline to induce more rapid mortality reduction, no attempt is made to do so. This is because of the desire for model B, like model A, to reflect currently conventional practice; and whereas the mortality-fertility relationship is widely discussed with respect to the population growth effects of faster mortality decline, the standard practice is not to adjust mortality assumptions in response to faster fertility decline when population projections are made.

As can be seen from table I-3, which presents the projection results, all projections start with a population of 10.0 million and extend for 100 years. The selection of the former figure is largely arbitrary; 100 years was chosen because it is the longest round period of time that would, at the higher rate of income growth used, allow the highest level of development achieved to fall within the range of values covered in table I-1.

As table I-3 of the text also shows, the application of the approach used produces implausibly large population totals toward the end of the projection period. This can probably be attributed at least in part to the well-known dangers of using cross-sectional relationships for projection purposes. Should the result of the cross-sectional approach be a failure to account for changes in technology leading to a structural change featuring lower fertility and (to a lesser degree) mortality relative to other development indicators over time, the population figures of table I-3 would overstate the amount of growth that could reasonably be anticipated. An alternate or supplementary possibility would be to view the large size of the population produced as a warning against relying on "normal" development to solve the population problems of poor countries, especially if slow progress is all that can be expected. For it seems clear that the population figures given are substantively unrealistic, even if technically correct, in the sense that progress toward them would in all likelihood be interrupted at some point by Malthusian checks and rising death rates that would lead to stabilization at some lower level.

Finally, it must be acknowledged that, despite the strenuous effort to disguise the fact, the projection approach used does at least some violence to the desire of McGranahan and his colleagues to take a "holistic approach" to development and to avoid any implication of causality among the variables they study. The projection approach is not so crude, certainly, as to attribute any causal superiority to per capita income; but it does require acceptance of the idea that different rates of overall development can be said to cause the different rates of fertility and mortality decline observed. This is contrary to the spirit of correspondence analysis by which the relationships of table I-1 were developed, being instead in the tradition of regression analysis which McGranahan and his colleagues chose not to apply.

The only defense to be raised against any objection raised on these grounds is that the transgression is not so much worse than standard practice as might at first seem to be the case. Whether established through correspondence or regression analysis, statistical relationships represent only associations, and those who would draw causal inferences about them do so at their peril. This being the case, it is not clear that one gains all that much more distance from causality by using a correspondence rather than a regression approach; or that the legitimacy of inferring causality from correspondence relationships is all that much less justifiable than the common practice of drawing causal inferences from regression results.

Table I-1: Correspondence Among Development Indicators

Indicators	Index Range										
	0	10	20	30	40	50	60	70	80	90	100
1. Crude birth rate	48.50	46.75	44.87	44.79	43.67	40.23	35.04	27.25	20.88	17.15	16.50
2. Infant mortality rate	161.70	148.60	135.67	125.87	106.84	86.32	64.35	45.47	32.48	21.52	13.86
3. Life expectancy	41.34	44.10	47.05	48.77	52.48	57.79	62.41	66.87	69.44	71.49	72.94
4. Animal protein consumption per capita per day	9.68	11.27	11.59	12.41	14.04	18.07	24.01	35.98	45.98	53.36	63.16
5. Percent literate, ages 15+	23.65	32.31	37.07	39.87	47.75	60.07	72.14	85.04	92.76	97.78	101.11
6. Percent aged 5-19 enrolled in primary and secondary school	23.50	26.80	31.08	35.67	43.76	50.69	56.49	63.27	69.33	75.15	80.68
7. Percent of dwellings with electricity	23.18	27.43	33.37	37.20	38.24	46.66	58.61	78.25	93.83	98.92	99.78
8. Newspaper circulation per 1,000 population	4.79	7.35	11.41	17.61	32.41	55.09	83.69	133.10	208.89	290.55	356.82
9. Telephones per 100,000 population	136.50	205.82	348.34	607.26	1,008.23	2,011.51	3,768.04	6,768.05	12,193.11	22,482.20	41,192.50
10. Automobiles per 1,000 population	1.88	3.21	4.81	7.05	8.58	13.22	23.07	46.12	92.52	166.79	299.65
11. Males in agriculture as percent of total male labor force	91.37	81.85	71.04	64.02	57.22	52.16	42.28	29.68	20.96	13.85	8.29
12. Steel consumption per capita (kg.)	3.68	5.66	8.63	14.85	26.67	43.56	73.67	143.68	275.05	442.57	571.69
13. Energy consumption per capita (kg.)	37.15	73.22	118.58	201.02	351.72	524.39	871.88	1,600.78	2,837.27	4,389.17	6,608.19
14. Percent GNP from manufacturing	9.79	10.16	10.46	11.62	14.47	17.92	19.49	22.22	27.18	30.54	32.26
15. GNP per capita (US \$)	72.00	90.29	117.95	163.10	230.46	304.09	464.77	727.58	1,081.07	1,675.49	2,500.47
16. Crude death rate	22.24	19.85	17.57	16.33	13.77	10.82	9.42	8.85	10.18	11.63	11.52
17. Population growth rate	26.26	26.90	27.30	28.46	29.90	29.41	25.62	18.40	10.70	5.52	4.98

Sources:

Lines 1-15 Prepared by D.V. McGranahan and colleagues at the United Nations Research Institute for Social Development, cited in W. Parker Mauldin and Bernard Berelson, "Conditions of Fertility Decline in Developing Countries, 1965-75," *Studies in Family Planning*, Vol. 9, No. 5 (May 1978), p. 109.

Lines 16-17 Estimated from lines 1 and 3 through the application of Coale-Demeny "West" family stable population tables.

Table I-2: Overall Development: Two Scenarios

YEAR	2% SCENARIO						3% SCENARIO					
	Per Capita Income	McGranahan Development Level	Crude Birth Rate	Life Expectancy at Birth	Infant Mortality Rate	Crude Death Rate	Per Capita Income	McGranahan Development Level	Crude Birth Rate	Life Expectancy at Birth	Infant Mortality Rate	Crude Death Rate
0	\$100	13.5	46.09	45.1	144	19.05	\$100	13.5	46.09	45.1	144	19.05
25	\$164	30.0	44.78	48.8	126	16.30	\$209	36.8	44.03	51.3	113	14.59
50	\$269	45.2	41.87	55.3	96	12.23	\$438	58.3	35.90	61.6	68	9.65
75	\$442	58.6	35.78	61.8	67	9.62	\$918	75.4	23.82	68.3	38	9.57
100	\$724	69.9	27.36	66.8	46	8.86	\$1922	93.0	16.96	71.9	19	11.60

Table I-3: Population Size (in Millions) under Different Overall Growth Scenarios: 3 Models

Year	2% Scenario	3% Scenario			
		Model A		Model B	Model C
		Crude	Adjusted		
0	10.0	10.0	10.0	10.0	10.0
25	19.7	19.9	19.8	19.6	19.9
50	40.5	44.2	42.4	37.6	40.7
75	82.0	93.0	87.5	62.1	67.6
100	142.9	160.7	151.8	81.0	85.9

Annex II: Income Distribution

The study of income distribution and demographic trends by T. P. Dyson, C. L. G. Bell, and R. H. Cassen, which serves as the basis for the income distribution illustration, is an extension of work on redistribution and growth initiated at the World Bank. The question posed concerns the mortality, fertility, and population growth effects of movement toward a more egalitarian pattern of income distribution.

Dyson, Bell, and Cassen explore this question through the use of a simulation model. It features a division of the population under observation into three groups by income: the top 20%, the middle 40%, the bottom 40%. The economy as a whole is assumed to grow at 5% annually from a starting average per capita income of \$100 (in 1970 dollars), with the gain distributed among the three income groups according to two alternate scenarios. Under the first, termed "constant shares," the income shares of the top, middle, and lower groups would remain fixed at their initial levels of 0.64, 0.28, and 0.08, respectively, typical of low income, high inequality countries. Under the second, called "changing shares," the income shares of the three groups would shift over time from the same initial distribution until, upon the attainment of a per capita income of \$300, they would approximate the pattern of an egalitarian Eastern European country, with the top 20% of the population receiving 33% of the national income, the middle 40% receiving 40%, and the bottom 40% receiving the remaining 27%. That distribution would remain constant thereafter. The level and distribution of national income thus determined define the per capita income of each income group of the population for each time period covered by the projection; and this per capita income serves as the basis for defining the fertility and mortality levels of each group. These fertility and mortality levels in turn determine each population group's growth rate; and the weighted sum of the growth experienced by the three groups represents the growth of the society as a whole. At the end of each projection period, people are redistributed among income groups so that the initial 20:40:40 size distribution is maintained despite differences in group-specific growth rates.

The relationship between per capita income on the one hand and fertility and mortality on the other is fixed by empirically derived equations of a curvilinear form. The simplest of the approaches taken, which are featured in the present example, are of two sorts. One is cross-sectional, based on international comparisons of income and fertility or mortality levels. The second is a time-series exercise, based on data from Taiwan. At low levels of income, the Taiwan time-series equations produce a significantly greater change in mortality and fertility per unit of income increase than do the international cross-sectional equations. The time series equations can thus conveniently be referred to as the "high response" equations, those produced by cross-sectional exercises as the "low response" equations.

The combination to be employed here for illustrative purposes combines the low response mortality equation of the cross-sectional analysis with the high response fertility equation of the Taiwan experience. This was selected

because, of the combinations for which the information presented in the published account of the Dyson-Cassen-Bell work is sufficient to support an exercise of the sort presented here, its results most closely approximate those produced by the more complex exercise whose outcome was thought by Dyson, Bell, and Cassen to offer the best reflection of reality. Over the range of income levels covered, the mortality equation describes a progressively declining amount of life expectancy increase per dollar of incremental income. The fertility equation features very slow fertility decline in response to income increase at low income levels, followed by a period of high response which slows as higher income levels are reached.

The summary mortality and fertility trajectories or variants for the society as a whole, expressed in terms of life expectancy at birth and the gross reproduction rate, resulting from the Dyson-Bell-Cassen exercise are supplemented by linear interpolation to provide a figure for each 5-year interval and are used as the bases for standard component-method population projections. The two fertility and two mortality variants produced in this manner (one fertility and one mortality variant from the "constant shares" scenario, one fertility and one mortality variant from the "changing shares" scenario) are presented in abridged form in table II-1. They provide the basis for four population projections, constructed in a manner analogous to those for the overall development illustration described in annex I. That is, the fertility and mortality trajectories of the "constant shares" scenario are used together in a baseline projection. The fertility variant of the baseline "constant shares" projection is combined with the mortality variant of the "changing shares" scenario and adjusted using the same procedure and correction factor described in annex I to produce a "model A" assessment of more equitable development's population growth implications. A combination of the "changing shares" fertility variant and the "constant shares" mortality variant results in a "model B" assessment. The "model C" assessment combines the "changing shares" mortality and fertility variants.

Each of the projections starts from the same arbitrarily selected population size of 10.0 million, as used in the overall development illustration; each employs the same initial gross reproduction rate (3.06) and life expectancy at birth (43.2 years), which is the weighted average of the initial values for those indicators used by Dyson, Bell, and Cassen for the upper, middle, and lower income groups; each projection starts with the same age distribution of the population, selected from the relevant Coale-Demeny "West" family stable population table; each employs the same age-specific fertility and mortality schedules; each projection is extended for the same 150-year span as in the original Dyson-Bell-Cassen exercise.

The resulting population size figures, presented in table II-2, are somewhat -- up to about 20% -- larger than those produced by the comparable Dyson-Bell-Cassen projections. This difference appears to result primarily from the use of single projections based on average mortality and fertility rates for the society as a whole, rather than the preparation and subsequent summing of parallel projections for each income group in the manner of Dyson, Bell, and Cassen.

Also, the population size figures, like those of the overall development illustration, all become implausibly large toward the end of the projection period. This suggests that they can best be regarded in the same light as suggested in annex I for the findings of the overall development exercise: as the inflated products of a failure to account adequately for technological change; and/or a warning against reliance upon an altered pattern of income distribution alone to avoid a Malthusian disaster.

Finally, it is to be remembered that the results presented in table II-2 relate directly only to the particular Dyson-Bell-Cassen exercise on which they are based. As noted, this is but one of several that Dyson, Bell, and Cassen undertook within the framework of their model. All of these exercises indicate that when both fertility and the mortality affects are taken into account (as in model C) the long-term result of more equitable income distribution in the "changing shares" projection is a population smaller than that of the baseline "constant shares" projection. In all but one of the exercises, the difference, measured in proportional terms, is greater than that of the exercise used here. Dyson, Bell, and Cassen do not produce the equivalent of "model A" or "model B" projections; but to judge from the way in which those projections are produced, it seems clear that a "model A" approach would result in a population slightly larger than that of the baseline projection in any exercise; and that a "model B" approach would inevitably yield a population at least somewhat and possibly significantly smaller than that resulting from the application of model C.

Table II-1: Income Distribution: Two Scenarios

Year	Constant Income Shares			Changing Income Shares		
	Per Capita Income	Life Expectancy	Gross Reproduction Rate	Per Capita Income	Life Expectancy	Gross Reproduction Rate
0	100	43.2	3.06	100	43.2	3.06
25	160	46.1	2.78	158	46.6	2.84
50	302	50.3	2.69	283	52.6	2.66
75	570	55.0	2.37	521	60.3	2.00
100	1,129	60.8	2.03	1,118	68.2	1.36
125	2,393	66.3	1.57	2,766	70.7	1.32
150	5,798	70.2	1.32	7,520	70.7	1.32

Table II-2: Population Size (in Millions) under Different
Income Distribution Scenarios: 3 Models

Year	Constant Shares	Changing Shares			
		Model A		Model B	Model C
		Crude	Adjusted		
0	10.0	10.0	10.0	10.0	10.0
25	18.5	18.5	18.5	18.6	18.7
50	34.0	34.5	34.2	34.6	35.2
75	63.0	66.8	64.9	61.9	65.7
100	111.3	126.2	118.8	94.1	106.4
125	182.4	219.1	200.8	124.9	149.7
150	266.0	328.0	297.0	155.4	191.4

Annex III: Child Spacing

The child-spacing illustration is based on a component-method population projection exercise, in which the age-specific distribution of fertility, the infant and child mortality rates, and the total fertility rate are varied to simulate the population growth effects of a change in the average interval between births.

The initial population size is set at the same, arbitrarily-established 10.0 million as in the previous illustrations. The initial principal demographic characteristics are those typical of the developing world as a whole in the late 1970s according to the standard figures of the United Nations: a total fertility rate just over 4.8; a life expectancy at birth of 55.0 years; and the age distribution consistent with these parameters estimated by the U.N. In the baseline projection, the total fertility rate and life expectancy are allowed to move toward an eventual low-level equilibrium at a rate approximately equal to that presented in the medium variant of the most recent United Nations projections. (The exact variants, like the values of the common initial demographic variables, are those used in the projections presented in Davidson R. Gwatkin and Sarah K. Brandel, "Life Expectancy and Population Growth in the Third World," Scientific American, Vol 246, No. 5 (May 1982), pp. 57-65.) Once reached, a net reproduction rate of unity is maintained in all projections for the remainder of the projection period, which is in each case extended for 125 years, until the achievement of a stable population.

As noted, the sources of variation between the projections arise from an extension in the length of the average birth interval, an extension which is posited for the purpose of this illustration as six months. All projections start with an assumed average birth interval of 24 months. In the baseline projection, this interval is maintained throughout the projection period. In the alternate scenario employed, the interval is assumed to lengthen to 30 months beginning with the second projection period and subsequently to remain at this level throughout. The assumption, in other words, is that it will be possible to lengthen the birth interval by six months within five years of the initiation of an effort to do so, and to maintain this gain into the indefinite future.

The selection of the 24- and 30-month intervals is largely arbitrary but not totally devoid of an empirical foundation. According to a summary of birth intervals based on World Fertility Survey data, the (unweighted) average interval for the nine countries surveyed was 28.2 months (with a range of from 24.4 to 32.0 months) in the early 1970s, up from an average of 26.2 months a decade earlier. (Calculated from data presented in German Rodriguez et al., "A Comparative Analysis of Birth Intervals," unpublished draft World Fertility Survey Technical Paper 2199, October 1983, Table 9.) An examination of these figures suggested that 30 months was the maximum interval length that could safely be used if one wished to be reasonably representative; the baseline 24 months is a bit low, but necessary to provide the change of six months thought to be the minimum required to produce interesting results, once the thirty-month ceiling is established. To suggest that such a lengthening might be

brought about within five years, implying a six-fold increase in the natural rate of lengthening, may be overly ambitious; but in view of the almost total lack of information about the effectiveness of interval-extension measures, it's not all that much worse than any alternate assumption; and handling it in this way simplifies the presentation. The choice of extending the average birth interval rather than reducing the number or proportion of births occurring below some specified interval -- which would be preferable should there exist some threshold level beneath which mortality rises sharply -- was made for reasons of computational ease.

This change in the average birth interval is assumed to affect mortality through a change in the infant and child mortality rate. It is assumed to influence fertility through changes in the inter-generational period and in the total fertility rate.

As for mortality, the effect of lengthening the average birth interval from 24 to 30 months is assumed to be an immediate 15% reduction of the infant and child mortality rate. In the 24-month variant of the mortality assumptions, the infant and child mortality rates used are those corresponding to the figures for life expectancy at age five that are common to all projections (a commonality which implies that mortality above age five is not affected by a change in interval length), as determined by the Coale-Demeny "West" family model life tables. In the 30-month variant, these infant mortality rates are reduced by 15%; and the child mortality rates are set, again by applying the Coale-Demeny "West" model life tables, at levels corresponding to the lower infant mortality rates.

The 15% figure was selected on the basis of a literature review that pointed toward an effect of somewhere between 10% and 20%. The review covered studies done for the World Health Organization, the World Fertility Survey, the World Bank, and the Population Council.

The WHO study included seventeen population groups in eight developing countries. On average, the ratio of mortality among infants born less than 24 months after the preceding offspring to that among infants born 24-47 months after was 1.93:1.00. If the length of the interval for those births occurring at less than 24 months is placed at 12 months and the average length of the 24-47 month interval at 36 months, and if the relationship between mortality and birth intervals is linear, such a ratio suggests that a six-month interval extension would produce about a 16% reduction in infant mortality. If the average of the 0-23 month interval is instead set at 18 months, *ceteris paribus*, the corresponding figure would be around 19%. (Calculated from figures presented in A.R. Omran and C.C. Standley, eds., Family Formation and Health: An International Collaborative Study in India, Iran, Lebanon, Philippines, and Turkey (Geneva: World Health Organization, 1976), pp. 216, 226, 235, 245, 255; and in A.R. Omran and C.C. Standley, eds., Further Studies on Family Formation Patterns and Health: An International Collaborative Study in Colombia, Egypt, Pakistan, and the Syrian Arab Republic (Geneva: World Health Organization, 1981), pp. 195, 206, 216.)

More recent work has suggested smaller differentials. The World Fertility Survey, for example, has found an average ratio of 0-23:24-47-month

interval infant mortality for the 29 developing countries covered of between 1.78:1.00 and 1.48:1.00, depending upon the estimation procedure used. (Calculated from figures presented in Shea Oscar Rutstein, Infant and Child Mortality: Levels, Trends, and Demographic Differentials, World Fertility Survey Comparative Studies, no. 24 (Voorburg, Netherlands and London, England: International Statistical Institute and World Fertility Survey, September 1983), p. 32.) The WHO differential would also seem larger than that suggested by the conclusion of a more sophisticated analysis of WFS data by James Trussell and Anne Pebley that "an improvement in birth spacing patterns, so that all births subsequent to the first are spaced at least two years apart" would reduce infant mortality by 10% and child mortality by 16% on average among the 25 countries studied. (James Trussell and Anne R. Pebley, "The Impact of Family Planning Programs on Infant, Child, and Maternal Mortality," draft background paper for the 1984 World Development Report of the World Bank, 31 October 1983, p. 20.) In addition, there are theoretical reasons for suspecting that oversimplifications in the analysis behind differentials such as those presented in the WHO study overstate their magnitude. (Beverly Winikoff, "The Effects of Birth Spacing on Child and Maternal Health," Studies in Family Planning, Vol. 14, No. 10 (October 1983), pp. 231-45.)

(The assumption that the differential used is applicable to all births rather than only to second and subsequent births is an obvious source of overstatement whose magnitude would vary inversely with the level of the total fertility rate. It would be offset to some unknown degree by the tendency for the preceding as well as succeeding births to be affected, which is not taken into account in the estimate presented. For evidence on this latter relationship, see John Hobcraft, John W. McDonald, and Shea Rutstein, "Child-Spacing Effects on Infant and Early Child Mortality," Population Index, Vol. 49, No. 4 (Winter 1983), pp. 585-618.)

Because of the many uncertainties surrounding the interval-mortality relationship, only limited confidence can be placed in any figure that might be selected. Fortunately this difficulty is not of major practical significance for the purpose of a projection exercise of the sort undertaken here since, as can be seen from the text, future population size is quite insensitive to variations in the strength of the relationship within the wide range of values that might reasonably be considered.

As indicated, the fertility consequences of lengthening the birth interval from 24 to 30 months are estimated in two ways. First, the changes in generational effects are estimated through the use of hypothetical age-specific fertility schedules that are altered to reflect a lengthening of the birth interval. Second, the influence of longer intervals on completed family size is estimated by adjusting the total fertility rate.

The hypothetical age-specific fertility schedules are designed in a way that sacrifices elegance for the sake of clarity. For simplicity, the same maternal age of first birth is adopted for all projections and held constant within each. The figure chosen, 26.0 years, is too high to be a reasonable approximation of reality but is adopted despite this shortcoming because of the reasonable levels of the mean age of birth produced by its use in connec-

tion with the other elements of the procedure applied. For the 24-month variants, the subsequent births are assumed to occur at 28.0 years, 30.0 years, and so on, with the fractional birth being allocated to the final interval. Thus, the 24-month fertility schedule for a total fertility rate of 2.50 would have one birth at 26.0 years, one at 28.0 years, 0.50 births at 30.0 years. The births under the 30-month schedule are analogously placed at 26.0 years, 28.5 years, 31.0 years, etc.

For computational purposes, these schedules are translated into the conventional format by allocating each birth between the two relevant age categories according to the birth's distance from each; and by summing the resulting figures. The procedure can be illustrated with reference to the just-mentioned 24-month variant with a total fertility rate of 2.50. Age 26.0 is 20% of the distance from 25 to 30. Accordingly, 20% of the birth at age 26.0 is allocated to age 30, 80% to age 25. Similarly, 40% of a birth at age 28.0 goes into age 30; 60% to age 25. The half-birth at 30.0 years is allocated totally to age 30. Adding these together produces an age-specific fertility schedule with 1.2 births at age 25, 1.3 births at age 30.

In the absence of any known empirical analysis of the relationship between birth interval length and total fertility, an arbitrary procedure is adopted. This consists of assuming that, at very high levels of fertility, an extension of the birth interval will result in an proportionate decline in total fertility; that, at low levels of fertility, an extension will produce no change at all in total fertility; and that the decline in the strength of the birth interval's influence on total fertility between these two points will be linear.

For the illustrative purpose at hand, the highest attainable or "natural" level of fertility is set at 10 births per woman at two-year intervals during a 20-year reproductive period. Assuming the length of the reproductive period to be fixed, an extension of the average birth interval from 2.0 to 2.5 years (i.e. from 24 to 30 months) would mean a reduction of the total fertility rate from 10 to 8 (20 years divided by 2.5 years/child), a decline of 20% from the initial figure. (For a discussion and presentation of this procedure, see John Bongaarts and Jane Menken, "The Supply of Children: A Critical Essay," The Population Council Center for Policy Studies Working Paper, No. 95 (April 1983), pp. 22-24.)

The level of fertility at which an extension of birth intervals would no longer exercise any effect at all on completed family size is assumed to be a total fertility rate of 2.0. This is roughly the fertility level needed for the population to replace itself when mortality is very low.

The assumption of a linear decline in the relationship between these two levels means that when the total fertility rate is at 6.0, the mid-point between them, a lengthening of the birth interval from 24 to 30 months would bring a 10% reduction in the total fertility rate. With the total fertility rate at 4.0, a similar birth interval extension would reduce the total fertility rate by 5%. And so on.

While this set of assumptions about the between the birth interval and the total fertility rate relationship seems as sensible as any other that might be imagined, it is strictly hypothetical in nature, as indicated earlier. To illustrate its influence on the projection results, the results of an alternate projection based on the extreme assumption that longer intervals produce no decline at all on the total fertility rate are also presented.

Table III-1 presents the mortality and fertility scenarios that result from the application of the assumptions described. Table III-2 contains the population figures produced by projections based on the scenarios in table III-1. The "model A - adjusted" figure incorporates the correction factor for the partially-offsetting fertility reduction caused by faster mortality decline, as described in annex I. The "model B - baseline TFR" and "model C - baseline TFR" population size estimates are developed on the basis of the assumption that longer birth intervals will have no effect on the total fertility rate. The "model B - adjusted TFR" and "model C - adjusted TFR" figures assume that the total fertility rate will be reduced by a lengthening of intervals in the manner just described.

Table III-1: Child Spacing: Two Scenarios

Year	24-Month Intervals			30-Month Intervals		
	Infant Mortality Rate	Life Expectancy at Age 5*	Total Fertility Rate**	Infant Mortality Rate	Life Expectancy at Age 5*	Total Fertility Rate***
0	94.08	58.40	4.818	94.08	58.40	4.818
25	48.14	64.32	3.096	40.92	64.32	3.011
50	12.20	71.34	2.080	11.28	71.34	2.080
75	11.28	71.60	2.080	11.28	71.60	2.080
00	11.28	71.60	2.080	11.28	71.60	2.080

*Average of separate male and female figures (with the relationship between them established by Coale-Demeny "West" family model life tables) used in projections.

**With an age-specific fertility schedule assuming 1st birth at mother's age of 26.0 years; 2nd birth at 28.0 years; 3rd birth at 30.0 years; etc.

***With an age-specific fertility schedule assuming 1st birth at mother's age of 26.0 years; 2nd birth at 28.5 years; 3rd birth at 31.0 years; etc.

Table III-2: Population Size (in Millions) under Different
Child-Spacing Scenarios: 3 Models

Year	24-Month Intervals	30-Month Intervals					
		Model A		Model B		Model C	
		Crude	Adjusted	Baseline TRF**	Adjusted TRF***	Baseline TRF**	Adjusted TRF***
0	10.00	10.00	10.00	10.00	10.00	10.00	10.00
25	16.03	16.15	16.09	15.88	15.60	16.00	15.72
50	21.77	22.05	21.91	21.45	20.88	21.73	21.14
75	25.85	26.26	26.06	25.39	24.55	25.80	24.94
100*	27.07	27.54	27.31	26.58	25.65	27.03	26.08

*Stable population

**Incorporating "24-Month" total fertility rate from table III-1

***Incorporating "30-Month" total fertility rate from table III-1

Footnotes

1. For clarity of presentation, the diagrams of figure 1 cover only the models' principal features. Hints of the many refinements and complexities that would need to be included in any fuller presentation appear from time to time in the text and the footnotes: the multiple channels between mortality and fertility change in models A and C for example, as noted briefly on page 4 of the text; the network of channels between the measures of model C and fertility or mortality changes, as suggested footnotes 7, 8 and 13; and the various ways in which fertility change can influence mortality change, as described in footnote 16.

2. For a sense of the issues covered and of the viewpoints presented in the extensive literature on this topic, see Samuel H. Preston, "Health Programs and Population Growth," Population and Development Review, Vol. 1, No. 2 (December 1975), pp. 189-99; and C. E. Taylor, J. S. Newman, and Narindar U. Kelly, "The Child Survival Hypothesis," Population Studies, Vol. 30, No. 7 (July 1976), pp. 263-78.

3. Among the many projection exercises following the time-honored technique of incorporating multiple fertility variants but only a single mortality variant are such sets of well-known international projections as those of the U.S. Census Bureau (for all the countries covered except China), the Community and Family Study Center (CFSC) of the University of Chicago, and Tomas Frejka. (The Census Bureau and CFSC projections are to be found in U.S., Council on Environmental Quality and Department of State, Entering the Twenty-First Century: The Global 2000 Report to the President, Vol. 2: The Technical Report, pp. 7-24 and pp. 24-38. The Frejka projections are in Tomas Frejka, The Future of Population Growth: Alternative Paths to Equilibrium (New York: John Wiley and Sons for the Population Council, 1973).

The well-established projections of the United Nations use multiple mortality as well as multiple fertility variants, but in a way that makes them even further removed from the spirit of the argument to be developed here than the multiple fertility/single mortality variant projections. That is, in the U.N. projections, rapid fertility and slow mortality decline variants are combined to produce a low population growth scenario; and the rapid mortality and slow fertility decline variants are used together to yield a high growth scenario. These produce estimates lying on either side of those constructed by use of the medium fertility and medium mortality decline variants. (A description of the method used in constructing recent U.N. projections appears in United Nations, Department of Economic and Social Affairs, World Population Prospects as Assessed in 1973 (ST/ESA/SER.A/60, "Population Studies No. 60"; New York: United Nations, 1977), pp. 7-13.) This is in distinct contrast to the theme appearing following pages that the nature of the relationships between fertility, mortality, and their determinants suggest that faster mortality and slower fertility decline, like slower mortality and faster fertility decline, are likely to be mutually inconsistent.

Of course, the desire to develop a set of realistic alternate visions of the future or to assess the population growth implications of faster fertility decline are not the only reasons why the projection exercises cited and the

many others like them were developed as they were. In at least some cases (notably that of Frejka), the desire was much less to approximate reality than to make a point through the use of assumptions that were in some cases explicitly recognized as unrealistic. In other cases, the use of multiple fertility variants resulted from a wish to acknowledge a lack of certainty about the rate of fertility decline implied by the mortality trajectory employed.

To the extent that purposes such as these lie behind the selection of variants used, the critique advanced here is not relevant. Amid a frequent lack of clarity about the reasons for selecting the particular configuration of variants used, however, are to be found strong hints that the desire to account for the possibility that fertility might decline more or less rapidly than suggested by the medium variant usually represents an important reason for using multiple fertility trajectories. It is in the many cases where this desire lies behind the selection of variants that the suitability of the multiple fertility/single mortality variant approach is questionable.

4. Examples of this tradition, also well established, are to be found in a wide range of presentations otherwise quite different in tone and orientation. For instance: Ansley J. Coale and Edgar M. Hoover, Population Growth and Economic Development in Low-Income Countries (Princeton, N.J.: Princeton University Press, 1958); presentations based on the "Rapid" model of the Futures Group (as in The Futures Group, "Central America: The Effects of Population on Economic and Social Development," in "Appendix to the Report of the National Bipartisan Commission on Central America" (March, 1984; mimeographed), pp. 541-98; and Julian L. Simon, The Economics of Population Growth (Princeton, N.J.: Princeton University Press, 1977). Since fertility and mortality are both exogenously determined in models such as these, there is nothing to prevent an alternation of mortality trends along with the changes of fertility trends introduced in the process of developing illustration based on them. This, however, is rarely if ever done; the convention, reflected in the publications cited, is to alter only fertility, leaving the path of mortality unchanged.

5. Examples include the fact that world population growth would continue for over 100 years and that population size would increase by over 50% even should universal replacement be immediately achieved (Frejka, The Future of Population Growth, p. 220); and the limited likelihood that the replacement effects of faster mortality decline would be large enough to offset fully the tendency toward faster population growth (Preston, "Health Programs and Population Growth;" Susan H. Cochrane and K. C. Zachariah, "Infant and Child Mortality as a Determinant of Fertility: The Policy Implications," World Bank Staff Working Papers, No. 556 (February 1983)).

6. Also, unlike models A and B, model C does not distinguish in advance between programs undertaken to reduce mortality and those designed to reduce fertility. Programs undertaken for either reason are assessed with the same framework, in recognition of the considerable overlap that can exist between mortality or fertility reduction measures, and of the resulting difficulty that can arise in identifying any given measure as primarily one or the other on a priori grounds. Such a distinction, if desired, would be made on the basis of a measure's observed effects rather than its stated objectives: that is, after determining the relative magnitudes of its mortality and fertility influence.

7. Or to be more accurate, a series or network of channels. For, as is to appear in the text and in the footnote that follows, it seems possible for a given mortality reduction measure to exercise more than one direct effect on fertility; for these effects to differ in direction as well as strength; and for the effect within each channel to differ in direction and/or strength at different levels of mortality, fertility, or the determinant(s) under consideration.

8. As will be seen, many of the conclusions of this paper rest on the proposition that the net direct fertility effect of most mortality reduction measures is, at least within the range of values under consideration, usually in the direction of a fertility decrease rather than a fertility increase. While this seems a defensible generalization based on the findings of the currently available empirical evidence, there is no obvious a priori reason why the overall direct fertility and mortality influence of a given measure need inevitably be in the same direction; and instances of possibly opposite mortality and fertility effects come readily to mind.

It is intuitively plausible, for example, that at least one effect of many health improvement measures might be an enhancement in reproductive capacity which, certeris paribus, would raise fertility; and there appear from time to time references to instances where such is thought to be the case. Reports of reduced birth weights and elevated levels of spontaneous abortion associated with malaria are common, for instance (B. J. Brabin, "Analysis of Malaria in Pregnancy in Africa," Bulletin of the World Health Organization, Vol. 61, No. 6 (1983), pp.1011-13; David J. Wyler, "Malaria: Resurgence, Resistance, and Research," Part I, The New England Journal of Medicine, Vol. 308, No. 15 (April 14, 1983), p. 877). Although it is not clear that the effect is large enough to be demographically significant, its existence suggests that malaria control measures would tend to raise fertility to at least some degree while lowering mortality. Another example concerns improved maternal nutrition, which some have suggested might increase fecundity and thus fertility at the same time that mortality is reduced. (Whether this actually happens is not clear: for a sceptical review of the literature, see John Bongaarts, "Does Malnutrition Affect Fecundity? A Summary of the Evidence," Science, Vol. 208, No. 4444 (May 9, 1980), pp. 564-69.) Also, the direct impact of education, especially male education in extremely poor rural societies, has upon occasion been found to be an increase in fertility (Susan Hill Cochrane, Fertility and Education: What Do We Really Know? World Bank Staff Occasional Papers, Number Twenty-Six (Baltimore: The John Hopkins University Press, 1979)).

Model C, which does not require that the effects delineated within it operate in the same direction, can be readily enough be used to assess the population growth implications of measures such as these; but the nature of the conclusion produced would obviously be quite different for a measure whose direct fertility effect is positive rather than negative. The existence of possibilities such as those just cited, even if infrequent or of modest quantitative significance in the settings under consideration, thus stands as an important warning against assuming that mortality reduction measures will inevitably result in fertility as well as in mortality decline.

9. For a review of the education-mortality evidence, see Susan H. Cochrane, Donald J. O'Hara, and Joanne Leslie, "The Effects on Education on Health,"

World Bank Staff Working Papers, No. 405 (July 1980), esp. pp. 56-92; and the papers presented at a 1981 symposium at the University of Michigan, as published in Health Policy and Education, Vol. 3, No. 1 (May 1982).

10. For a much fuller review of these and the other mechanisms involved, see Cochrane, Fertility and Education.

11. A discussion of these anti-malaria approaches is to be found in Gordon W. Harrison, Mosquitoes, Malaria, and Man: A History of the Hostilities since 1880 (New York: E. P. Dutton, 1978). For a description of the techniques used against smallpox, see Donald A. Henderson, "The Eradication of Smallpox," Scientific American, Vol. 235, No. 4 (October 1976), pp. 25-33.

12. A direct impact on mortality would be difficult to envision for a family planning service program providing only contraceptive supplies, for example.

13. As with the direct fertility impact of mortality reduction measures described in footnote 7, what's involved is often a series of direct mortality effects rather than a single effect. And while the net impact of these direct effects usually seems to be a reduction rather than an increase in mortality, there is no basis for supposing that all effects will operate in such a manner or that the balance among them will inevitably lie in this direction, for reasons analogous to those discussed in footnote 8 which argue against the assumption that the direct mortality effect of fertility reduction measures will inevitably be a decline in death rates.

The point can be illustrated with respect to the infant mortality impact of maternal employment outside the home, an example of an often-advocated approach to fertility reduction. This is a case where countervailing direct mortality effects seem to exist, and the relative strengths of the competing forces remains uncertain. On the one hand, there is the beneficial effect of the extra income, an effect strengthened by suggestions that any incremental income earned by mothers is more likely to be spent in ways conducive to improved child welfare than the same incremental income earned and controlled by men. (See, for instance, the studies cited in Daisy Hilse Dwyer, "Women and Income in the Third World: Implications for Policy," The Population Council International Programs Working Papers, No. 18 (June 1983), pp. 6-7.) On the other hand, some argue that maternal nurturing and stimulation are central child development, implying that extra-household employment, by reducing the time and inclination of mothers to care for their children, can contribute to increased infant mortality. (A review of the literature in this tradition is to be found in footnotes 2-4 of Barry M. Popkin, "Rural Women, Work, and Child Welfare in the Philippines," in Women and Poverty in the Third World, ed. Mayra Buvinic, Margaret A. Lycette, and William Paul McGreevey (Baltimore and London: The Johns Hopkins University Press, 1983, p. 174.) It seems possible for both forces to exist simultaneously, with one being stronger in some cases, the other in others. If so, the well-established association between extra-household maternal employment and lower fertility (*ibid.*, footnote 1, p. 174) would suggest such employment as an instance of a fertility reduction measure resulting at least upon occasion in higher rather than lower infant mortality.

14. See, for example, the material presented in Robert Repetto, Economic Equality and Fertility in Developing Countries (Baltimore and London: The

John Hopkins University Press for Resources for the Future, 1979). An alternate view is to be found in Bryan L. Boulier, "Income Distribution and Fertility Decline: A Sceptical View," Population and Development Review, Vol. 8, Supplement (1982), pp. 159-78.

15. Particularly well known in this respect is the relationship between income increase and food purchase embodied in "Engel's Law," based on the finding that the income elasticity of demand for food is normally well below unity, so that the proportion of family income spent on food declines as income rises.

16. Fertility declines of this nature appear to be the norm, as older, high-parity women predominate among the early acceptors of contraception (Dorothy Nortman, "Changing Contraceptive Patterns: A Global Perspective," Population Bulletin, Vol. 32, No. 3 (August 1977), pp. 20-22). This suggests that family planning programs usually result in a reduction in infant and child mortality, at least in their early stages.

Other patterns are possible, however. A recent report from Costa Rica suggests that fertility decline there began among younger, better-educated women (J. Mayone Stycos, "The Decline of Fertility in Costa Rica: Literacy, Modernization and Family Planning," Population Studies, Vol. 36, No. 3 (March 1982), p. 24). In such a case, the effect of the fertility decline per se (although not necessarily the overall mortality impact of the education producing it) could well be a rise in the infant mortality rate, since the likelihood of death of those infants whose births were initially averted would presumably have been lower than average in view of their prospective mothers' age and educational status.

It is, of course, possible to question whether changes of this sort in the infant mortality rate, whether rises or falls, are of much significance for welfare. As suggested by the careful way in which the text is worded, there would seem a legitimate distinction between changes in the infant mortality rate that result from: a) a reduction in the number of births in a higher- or lower-than average risk category that do not affect the death probabilities of those infants whose births are not prevented; and b) a reduction in fertility that leads to a change in the death probabilities experienced by those born. An example of the former, under discussion here, would be the prevention of the final birth of an older, high-parity woman. An instance of the second would be a delay in the age of first birth or a lengthening of birth intervals. If welfare improvements are defined as improvements in the survival probabilities of those born, then much of the infant mortality decline attributable to fertility reduction would be irrelevant for welfare (although not for population growth).

Be this as it may, what has been said indicates that the indirect mortality impact of fertility reduction measures cannot safely be considered fixed. It will, rather, vary according to the pattern of fertility reduction produced by the measures under consideration. In this respect, it would appear to differ from its counterpart -- the indirect fertility effect of mortality reduction measures -- whose strength, it was claimed earlier in the text, is likely to be the same whatever the measure producing the mortality decline in question. (A lucid discussion of the issues involved in the

assessment on family planning's impact on infant and child mortality, which served as a starting point for many of the thoughts just expressed, appears in James Trussell and Anne R. Pebley, "The Impact of Family Planning Programs on Infant, Child, and Maternal Mortality," draft background paper for the 1984 World Development Report of the World Bank, 31 October 1983.)

17. The principal source document on primary health care is the "Declaration of Alma-Ata," an outcome of a September 1978 international conference in that city sponsored by WHO and UNICEF. The report on this conference, which includes the declaration, is available as World Health Organization, Alma-Ata 1978: Primary Health Care, WHO "Health for All" series, No. 1. The fullest and most recent description of the UNICEF "child survival revolution" appears in James P. Grant, The State of The World's Children 1984 (Oxford: Oxford University Press, 1983). For the outlines of the Ford Foundation's approach, see The Ford Foundation, Child Survival/Fair Start, Working Paper from the Ford Foundation (New York: The Ford Foundation, August 1983).

18. When originally presented, in the 1982-83 State of the World's Children report, the program consisted of six measures and was known as "GOBI-FF." The third "F," representing female education, is a later addition.

19. Grant, State of the World's Children 1984, p. 57. (Pp. 25-60 of this document contain a fuller description of the seven GOBI-FFF measures.)

20. Even the immediate virtual elimination of infant and child mortality would produce a population less than 10% larger at stabilization in the typical developing country than the population associated with a more normal trajectory -- a difference far smaller than the near-trebling that would occur even were mortality not to decline more rapidly. Even this would overstate the size of the true difference, since the larger population size would have to be reduced by adjusting for the faster mortality decline's influence on fertility which, even if not fully offsetting, could still be important. (Davidson R. Gwatkin and Sarah K. Brandel, "Life Expectancy and Population Growth in the Third World," Scientific American, Vol. 246, No. 5 (May 1982), pp. 63-65.) Also, as even the most enthusiastic proponents of GOBI-FFF programs would be quick to point out, the amount of extra mortality decline such programs produce will inevitably be much less than that of the extreme case presented here.

21. Grant, State of the World's Children 1984, pp. 25-60 passim. For other recent work highlighting the role of personal behavior and belief, see W. Henry Mosley, "Will Primary Health Care Reduce Infant and Child Mortality? A Critique of Some Current Strategies, with Special Reference to Africa and Asia," paper prepared for the IUSSP Seminar on Social policy, Health Policy and Mortality Prospects, Paris, 28 February - 4 March 1983 (mimeographed); and J. C. Caldwell, P. H. Reddy, and Pat Caldwell, "The Social Component of Mortality Decline: An Investigation in South India Employing Alternative Methodologies," Population Studies, Vol. 37, No. 2 (July 1983), pp. 185-205.

22. James E. Kocher, Rural Development, Income Distribution, and Fertility Decline, An Occasional Paper of the Population Council (New York: The Population Council, 1973); Robert S. McNamara, Address to the Massachusetts Institute of Technology, Cambridge, Massachusetts, April 28, 1977 (Washington:

The World Bank, 1977); William Rich, Smaller Families through Social and Economic Progress, Overseas Development Council Monograph No. 7 (Washington, D.C.: Overseas Development Council, January 1973). A review and citations of material presenting the different positions on development's contribution to population are to be found in Bernard Berelson, The Great Debate on Population Policy: An Instructive Entertainment, An Occasional Paper of the Population Council (New York: The Population Council, 1975).

23. In the sense that a reduction of, say, half a percentage point in the crude birth rate would result in a decline of half a percentage point in the population growth rate.

24. McNamara, Address to the Massachusetts Institute of Technology.

25. Ibid, p. 3.

26. Ibid, p. 28.

27. Whether McNamara himself assumed this is not clear. On the one hand, as indicated in the text, McNamara did not try to provide an explicit estimate of the amount of reduction in population growth that might result from the implementation of the measures he recommended. On the other hand, he preceded and set the stage for his discussion of these measures with a presentation of figures by Tomas Frejka demonstrating the growth benefits of achieving a net reproduction rate of unity in 2000 rather than 2020 A.D. -- figures which, as noted in footnote 3, were prepared by means of a "model B" approach that allowed the fertility but not the mortality assumption to vary. (Ibid., pp. 10-11.)

28. Examples include Hollis Chenery and Moises Syrquin, Patterns of Development, 1950-70, A World Bank Research Publication (London: Oxford University Press for the World Bank, 1975); D.V. McGranahan et al., Contents and Measurement of Socioeconomic Development: A Staff Study of the United Nations Research Institute for Social Development, Praeger Special Studies in International Economics and Development (New York, Washington, London: Praeger Publishers, 1972); and, for Latin America and the Caribbean, Frank William Oechsli and Dudley Kirk, "Modernization and the Demographic Transition in Latin America and the Caribbean," Economic Development and Cultural Change, Vol. 23, No. 5 (April 1975), pp. 391-419.

29. McGranahan et al., Contents and Measurement of Socioeconomic Development.

30. T. P. Dyson, C. L. G. Bell, and R. H. Cassen, "Fertility, Mortality, and Income -- Changes over the Long Run: Some Simulation Experiments," The Journal of Development Studies, Vol. 14, No. 4 (July 1978), pp. 40-78.

31. Through policies and programs, in other words, operating on two of the four segments into which birth intervals are frequently divided for analytical purposes: i.e. on postpartum infecundability and on conception delay. The third and fourth segments -- time added by spontaneous intrauterine mortality, and by gestation -- are taken as notably less amenable to policy or program intervention. (For a fuller presentation of this four-segment categorization, see John Bongaarts and Jane Menken, "The Supply of Children: A Critical

Essay," Population Council Center for Policy Studies Working Paper No. 95 (April 1983), pp. 20-21.)

32. In interpreting this result, the importance of the assumption employed concerning the influence of longer birth intervals on total fertility is to be kept in mind. The result presented incorporates the assumption that a shift from 24- to 30-month intervals would reduce the total fertility rate somewhat, in the manner described in annex III. Were one to assume instead that the birth interval had no influence on total fertility, the "model C" population would be almost identical to that of the baseline. The inter-generational effects of birth interval shift under discussion, in other words, are large enough to offset fully the tendency toward faster growth resulting from the reduced infant mortality produced by that shift; the magnitude of any reduction in population size relative to that on the baseline resulting from longer intervals will depend on the strength of the mortality-total fertility relationship.

33. Such as malaria in sub-Saharan Africa. See footnote 8.

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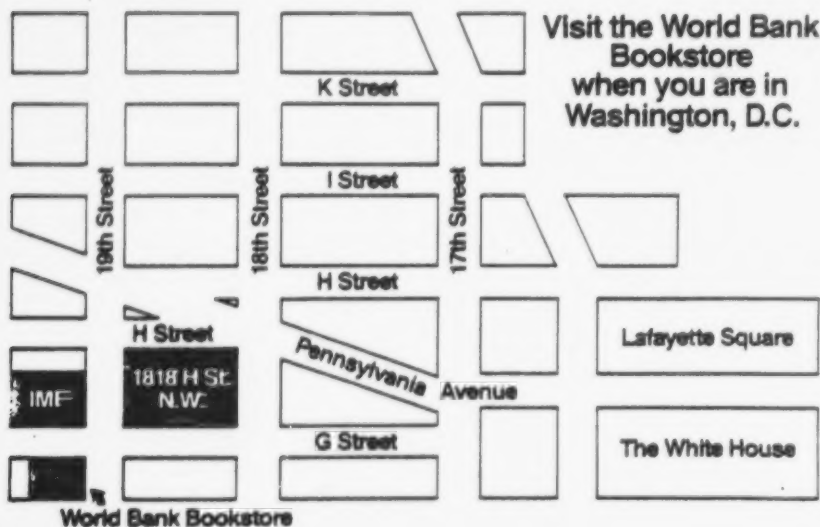
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